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VETERINARY MEDICINE

APRIL 1943

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No. 4

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Achievements of 1942

Efficient U. S. Army system of evacuating the wounded under fire, sulfanilamide and blood plasma banks scored a notable triumph in saving lives at the Pearl Harbor raid, where almost 100% of abdominal wounds healed without infection, less than 4% of compound fractures and flesh injuries became infected.

Propylene glycol vapor was found to be a safe, effective substance for destroying influenza virus and other germs in the air in laboratory experiments and was credited with reducing significantly respiratory infections among 16 children in the ward of an institution where it was tried.

Discovery of chemicals, probably enzymes, in the bodies of young mice which destroy the drug-resistant waxy parts of the tuberculosis germ is announced. There is hope of the development of an enzyme preparation that may be used in the treatment of tuberculosis.

Hope for a chemical conquest of tuberculosis was encouraged by announcement of successful results in treatment of patients with a relatively new drug, Promin, and by announcement of even greater success in treatment of tuberculous animals with a related chemical, di-amino-di-phenyl sulfone.

First, and strikingly successful, use of the Moorhead Foreign-Body Finder, in which a radio frequency circuit with movable coil and steel finger detects shell fragments and the like in war wounds within a few minutes instead of hours as by X-ray and probe, was reported from Pearl Harbor.—*Science.*

While all Christendom bent its energies (1914-18) to the killing of young men, nature outstripped the statesmen; influenza killed more victims in a few months than did all the armies in four years. Estimates of the world-wide mortality run from 10 to 21 million.—*Plague on Us.*

Peace Only with Honor*

We love peace, as we abhor pusilanimity; but not peace at any price. There is a peace more destructive of the manhood of the living man than war is destructive of his material body. Chains are worse than bayonets.—*Douglas Jerrold.*

Among the new developments in drugs and technics, I speak particularly of the sulfa compounds that have proved themselves miracle medicines in checking otherwise fatal infections. Blood plasma has saved innumerable lives that would have been lost under the circumstances that prevailed during World War I.—Rear Admiral John Downs, U. S. N.

* Reprinted from *Veterinary Medicine*, August, 1918.

Swine Need Vitamin B_6

That deficiency in hogs of pyridoxine (vitamin B_6) causes loss of appetite, poor growth, fits and anemia has been discovered by scientists of the University of California College of Agriculture. Very minute daily doses (five milligrams of pyridoxine per 100 pounds body weight) restored the swine to normal health.

Swine Pox

Swine pox is not usually a serious disease in itself, but it predisposes to other diseases and can be a complicating factor when other infections are present. Furthermore, pox occurs under conditions which favor or foster other infectious and parasitic diseases. The occurrence of swine pox to any appreciable extent in the herd may therefore, be taken as a warning that all is not well. Pox is spread from pig to pig principally by the hog louse; widespread pox usually goes hand in hand with a relatively heavy louse infestation. Control of lice is essential in limiting the spread of swine pox. Hog-cholera virus should not be employed as an immunizing agent in herds showing extensive pox.—Department of Animal Pathology and Hygiene, University of Illinois, Urbana.

Fertility and Hatchability Confused

Fertility should not be confused with hatchability. Fertility for the entire lot of eggs may be low and yet high hatchability of the few fertile eggs may prevail. Low hatchability might prevail in a lot of eggs that showed high fertility. There are interlocking relationships between the two, but the fundamental factors which govern fertility differ from those which control hatchability.

There has been considerable discussion as to whether the use of light and shelter will reduce fertility and hatchability. Where proper conditions are provided and the birds are in good physical condition, light and shelter seem to have little influence on fertility and hatchability.—*Bennion, N. L.*, in *Poultry Digest*.

Cottonseed meal and peanut meal are approximately equal in protein content—43%. A test at the Texas Agricultural Experiment Station showed them to be of equal feeding value in the ration of fattening steers.

In an experiment at the West Virginia station, calves creep-fed a grain mixture, while running with their dams on good pasture, were ready for market four months earlier than calves not creep-fed and fattened in a dry lot after weaning.

Treatment of Mastitis

Udder infusion, striking at the seat of the trouble, in the treatment of mastitis has long intrigued the practitioner. A survey of the literature reveals that a large number of agents have been used in this manner with indifferent results. It was only a score of years that research workers began investigations in this field and not till 1929 that material progress was made in finding agents effective against udder infection and not, at the same time, permanently injurious to the parenchymatous tissue of the organ.

For a decade various of the dyes were used for the injections—rivanol and entozon (both of which are acridine derivatives) and ubersan, acriflavine and trypaflavine being the agents most used. Of these the latter made the best record.

Trypaflavine (1:1500) in a 20% glucose solution gave 90% of cures in mastitis due to the *Str. agalactiae*. The use of the hypertonic sugar solution lessens absorption of the bactericide and it was found that in 98% of the cases the total amount of the infusion can be milked out up to 10 minutes after it is injected.

In 1939 and since, Dubos, Klein, Little, Bryan, Palmer and others have experimented with three medicaments of a different type — gramicidin (tyrothricin — a product *B. brevis*), novoxil, a colloidal suspension of silver oxide in mineral oil and sulvetil, a suspension of sulfanilamide in light mineral oil. With all three of these agents the results were better than those obtained earlier from the dyes.

Vitamin C in the War

It is known that Germany has been using vitamins for her fighting forces and depriving conquered populations of sources of vitamin B_1. Great Britain, too, has been alert to the value of vitamins A, B_1 and C as aids to the war effort. Our armed forces are supplied not only with a vitamin-rich diet but with supplemental vitamins also. This is especially true of synthetic vitamin C, ascorbic acid, of which the United States produced 17 tons in 1941 and 100 tons in 1942, much of which was sent to our Allies.

Vitamin C is destroyed by infection and by a number of industrial and military poisons. It is also lost in appreciable quantities in heavy perspiration. One important industrial organization adapted the practice of giving each worker, daily, a tablet containing vitamins C, B_1 and common salt. Improvement in general vigor was most encouraging, there being no more heat prostrations. Similar conditions often exist in war areas in the tropics and in North Africa. The function of the vitamin C may go beyond mere replacement of the amount lost. It may combat heat shock.

Shock results form a number of causes, so vitamin C therapy should be considered. It was found that guinea pigs injured to an extent that otherwise would have caused 100% mortality survived when given considerable vitamin C immediately after injury (subcutaneous injection). Delay of one hour reduced recovery to 50%. This treatment is being tried on humans in a few medical centers and might well be tested by surgeons in our armed forces. Numerous investigators have reported a decrease in the ascorbic acid concentration of the blood plasma following operations.

Rapid healing of wounds is a matter of military concern, so it might well be noted that vitamin C plays an important role in healing of fractures and of wounds in general. The health of intercellular tissue depends, in part, upon an adequate level of this vitamin.

A confidential report from a T.N.T. plant in Britain strongly endorses the vitamin C treatment for workers. From tests, the physician in charge firmly believes that T.N.T. destroys vitamin C. As treatment the poisoned workers were given 100mg daily of vitamin C, "each dose being followed by one drachm of bicarbonate of soda in solution."

Benzene and toluene are industrial solvents of importance to war industries as well as basic materials for the manufacture of T.N.T. and other explosives. They are somewhat toxic in liquid or vapor form. It is reported that dust of lead or its compounds destroyed some vitamin C in the bodies of workers exposed to the toxic dust. And zinc oxide fumes given off when brass is melted causes symptoms that are somewhat reminiscent of lead poisoning. Daily doses of 200mg vitamin C resulted in great improvement of health in most cases.

To complete this list of items of military value, it might be mentioned that 100 cases of insomnia treated with vitamin C showed excellent improvement. It would seem that both C and B_1 could be useful in many cases of nervous disturbance.—*Science*.

Uninspected Slaughter of Food Animals Increasing

The slaughter of hogs at plants where federal inspection is maintained fell two million short of the estimated slaughter for November and December 1942. A large part of this deficit is accounted for by the increased slaughter on farms and in locker plants where there is no inspection. Both of these slaughters, particularly the former, are important sources of supply for the black market.

J. S. Hoffman, president of the Washington Federation of Butchers, states that 10% of the meat used in the State of Washington is sold through "non-authorized", i.e. bootleg sources. B. V. Urwin, secretary-treasurer of the Detroit Independent Meat Packers Association declares Detroit's supply of meat "continues bad and is getting worse", and not only are farmers within private trucking distances slaughtering their own animals for the black market but that 25% of the animals shipped to the Detroit stock yards go, not to the packing houses, but to nearby farms for slaughter and thence to Detroit's unlicensed and unregulated market. The Detroit Board of Health has obtained a ruling recently allowing it to seize all uninspected meats offered for sale, but the number of inspectors that it has is inadequate to cope effectively with the black market.

A committee of the Eastern Independent Packers, after a survey of the situation in New England, reports that "there has been so much bootlegging of meat that the legitimate dealer is being forced out of business."

The quality of meat on the black market is fast declining to the quality of bootleg liquor during prohibition days. Diseased animals are slaughtered along with the fit in makeshift establishments without even the most primitive sanitary facilities and retailed from unlicensed markets that often are indescribably filthy. Further, in this trade, the conscience, if, any of the bootleggers is the principal safeguard against the sale of the flesh of "cold slaughtered" animals.

Taking in Lots of Territory

He (the dog owner) might very nearly as well attempt to compete with Chrysler, Ford and General Motors in building his own automobile as to produce his own dog feed in competition with the leading dog food manufacturers of today.

* * * *

A bill for the control of artificial insemination is being considered by the House of Commons (England). If it is acted upon favorably, the Minister of Agriculture will be authorized to make regulations to control the practice of artificial insemination and to prohibit (subject to exemptions) the distribution and sale of semen of any animal except under license. These regulations may apply to cattle, sheep, goats, swine, horses and domestic fowl. Under the regulations licenses will be required for the importation or exportation of semen.—*Vet. Rec.*, Dec. 26, '42.

* * * *

Wounds at Pearl Harbor

Between 7:50 a.m. and 1:50 p.m., Dec. 7, 1941, 400 major casualties were received by the surgical service at Tripler General Hospital, Fort Schafter, Honolulu. Infections developed in less than 1% of the large number of wounds treated—apparently convincing proof of the effectiveness of the routine procedure in wound treatment. The procedure is based on the following fundamental precepts:

"1. No person in shock should be subjected to surgery except for control of hemorrhage. . . .
2. All accidental wounds are infected and should be treated by:
   (a) Thorough cleansing with soap and water, soap and water, and more soap and water.
   (b) Thorough debridement, with removal of all devitalized tissue until there is normal appearance of tissue; bleeding; contraction (in the case of muscle).
   (c) The entire wound should be covered with sulfanilamide powder, or alternatively by sulfathiazole powder.
   (d) No wound should be closed. Stitches may be placed, to be tied at the end of the third day if no infection is present; or the wound may be left without sutures."

The B. A. I. Loses Meat Inspection

February 9th, Secretary Claude R. Wickard, United States Department of Agriculture, announced an order transferring the Meat Inspection Division of the Bureau of Animal Industry to the Livestock and Meats Branch of the Food Distribution Administration. Probably the only gratifying statement in connection with the order is: "The present administrative policies of the meat inspection service will be continued without any lowering of the high standards of inspection already established." It will be noted there is no promise of improvement.

Removing meat inspection from the Bureau is a poor reward for a remarkably high class, efficient service that has been of great value to public health and to the livestock and meat processing industries. More than any other factor, federal meat inspection has inspired confidence in the wholesomeness of market meat and has increased its use by the public and thereby improved the level of nutrition of the general population. It has in the same measure contributed to fair practices in the meat trade, and to an increased market, both domestic and foreign, for the livestock industry. In the more than half-century that the federal meat inspection service has been in operation there has been but a single breath of scandal leveled at it—"The Jungle." An official investigation completely exonerated the Bureau in that instance.

Much objection to the transfer was apparent, immediately upon the announcement of the order of February 9th and execution of it was delayed. However at the present writing the probabilities are the Secretary will not withdraw the order. A discussion of the causes that brought about this break in the ranks of the Bureau or rather which permitted it to occur can be of value, only as it may serve to avoid further emasculation now that the size and strength of the organization has been lessened and its prestige lowered. Five years ago, with its long record of unimpeachable meat inspection, its unequaled record in disease control and eradication, its brilliant record in biological research and discovery and particularly its success in bovine tuberculosis eradication, no Secretary of Agriculture would have had the temerity to assault the Bureau. In recent years two measures—opposition to the importation of Argentine beef and the brucellosis eradication project—have cost the Bureau many friends in high places and earned for it not a few influential enemies. Perhaps in the aggregate these were enough to tip the balance to the side of "divide and conquer."

As the chief federal guardian of animal health in this country the Bureau was obligated to concern itself with the question of importation of Argentine beef. But perhaps it would have been better to have rendered an opinion based upon the best scientific information it could assemble, issued a warning and remained detached from the heated controversy which was always about 99% political and commercial and 1% sanitary.

The brucellosis eradication project has been even more costly in friends and prestige. When the project was launched in 1934, this magazine counseled the veterinary profession not to forget, nor to let it be forgotten by the livestock industry, that the project was a cattle reduction and not a disease control project. It was pointed out repeatedly that the elimination from our dairy herds of thousands of heavy producing, regular breeding cows, that possessed an immunity or high resistance to the Brucella organism, must inevitably exact retribution and that, until a biological or other test was developed that could discriminate between infected and immune animals, no test should be made the sole arbiter of life or death for valuable animals. Further, a delay in giving official recognition to vaccination was costly in prestige with dairy press and with the dairy industry.

With maximum employment of vaccination and ceasing the slaughter of high producing, regular breeding cows, a good deal of credit can yet be salvaged from the brucellosis control project.

A British Veterinary Educational Trust

The *Times*, London, writes editorially of the trust sponsored by Dr. W. R. Wooldridge, president of the British National Veterinary Medical Association:

"On many occasions since the last war attention has been drawn in these columns to the strange anomaly that, while breeding and exporting some of the best livestock in the world, Great Britain has lagged far behind other countries in the matter of equipment for the study and teaching of veterinary science. As long ago as 1929 a departmental committee appointed by the Minister of Agriculture strongly criticized the condition of the Royal Veterinary College. In the following year Lord Harewood raised the question in the House of Lords. Eventually a royal charter was granted constituting a new governing body, and in 1937 the present buildings of the college (replacing those that had been in use since 1791) were opened by the King and Queen. All this showed distinct, though slow, progress; yet it was not enough. Another government committee, reporting in 1938, declared that 'veterinary education has been starved, the veterinary schools are overcrowded, teaching staffs are inadequate . . . facilities for clinical and practical training are insufficient and the system of education and courses of study also need amendment.' These were strong criticisms, which the committee supplemented with valuable suggestions. One of which was that each veterinary school should have its field station, and in some of them—the Liverpool Veterinary College, for example—this has now been achieved. Obviously, however, there can be no complacency over a state of affairs which has lately drawn from such an authority as Sir Arthur Olver the accusation that 'there is no other country in which livestock has such tremendous importance or in which so little has been done for veterinary education. . . . The necessary facilities are still not available in this country for adequate practical instruction.'

"With these facts in mind it is possible to appreciate the full importance of the announcement made by the president of the National Veterinary Medical Association that a veterinary educational trust has been formed with the object of improving veterinary education in this country and in the hope of raising for that purpose a fund of at least a million pounds. The sum is large, but it will give the nation an idea of the importance of the issues at stake. There are in this country, as Dr. Woolridge has reminded us, only 2,000 active veterinary surgeons to cope with a task that could well employ twice that number. Britain's inadequate educational services, moreover, have the responsibility of providing veterinary surgeons for the Colonial Empire. The war has shown up some deficiencies here, as in other places. Animal health is an essential part of the economy of husbandry. Animal diseases must mean loss and waste and may, in some forms, have their effect upon public health. The revival of British agriculture on a permanent basis will demand all the aid that science can give and, not least, all that an improved and developing system of veterinary science can contribute in the way of prevention as well as of cure. More veterinary surgeons and a better training are needed, and a million pounds is by no means too large an endowment to demand for these purposes."

* * * *

Feeding Problems in War

Feeding cattle for winter milk is one of the big problems of the war. Roots, silage, cabbage or sugar-beet tops and hay should be fed after milking. Feed concentrates before milking. Do not give hay or straw just before or during milking, because of their dusty nature.—*Ministry of Agri. England.*

* * * *

The principal export of Nigeria (at least, the principal export of animal origin) has long been cattle hides and goat and sheep skins. In normal times it amounted to some five million dollars annually. For years this trade suffered heavy losses from damage caused by bad flaying and faulty methods of drying. An improvement scheme under the control of the veterinary department in eight years went far to curing the faults and largely increased the value of the hides and skins in the export market.

* * * *

Iraq's earliest known inhabitants, the Sumerians, were fond of pork. They also ate a good deal of mutton and beef, and smaller quantities of game and fish. These facts are brought out in a study of animal bones found in buried city ruins at Tell Asmar, a site near the Tigris River a few miles from Baghdad. The study was made for the Oriental Institute of the University of Chicago by Dr. Max Hilzheimer, noted German zoologist. The most interesting thing about the finds is the high proportion of pigs. You couldn't sell pork for a cent a ton in that neighborhood now; the inhabitants are all Mohammedans.—*S. N. L.*

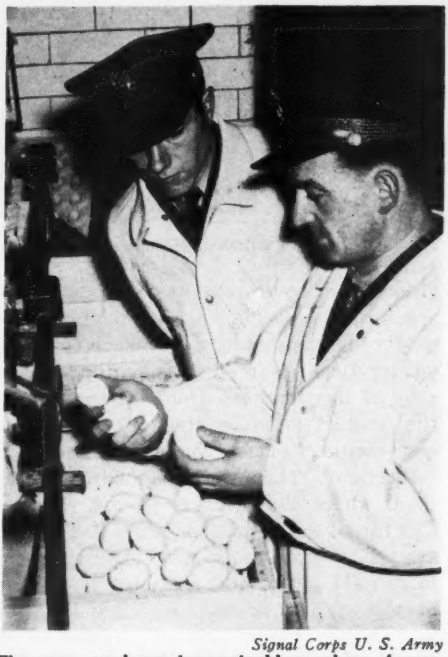
Inspection of Meats for the United States Army

By LIEUT. COL. FRED C. WATERS, V.C.,
Depot Veterinarian, Chicago Quartermaster Depot

OFFICERS and enlisted men of the Veterinary Corps stationed at the Chicago Quartermaster Depot are responsible for the inspection of meat products prepared in Chicago to be consumed by the army. Of course this work has expanded greatly since the inception of the Defense Program and still more since the outbreak of war. A staff of 72 veterinary officers and 80 enlisted men is required to handle the contracts which are being awarded every day.

Any establishment dealing in the products the army needs is allowed to bid on Government contracts provided certain sanitary requirements are maintained. Packing houses which satisfactorily pass the inspection of the federal Bureau of Animal Industry are allowed to supply the army without further question as to sanitary conditions. In the event dairy products, fish, or poultry are to be furnished, the army makes its own sanitary inspection, which is conducted by a veterinary officer. The sanitary conditions which require investigation include suitability of site and surroundings —that is, the general cleanliness of the location, adequacy of water supply and draining facilities; the interior construction of the building—whether it is constructed of suitable material and is in good repair; ventilation; lighting; cleanliness and condition of equipment and utensils; disposal of by-products; and the handling, packing and transporting of the products. The employees of the plant are also subject to scrutiny in regard to cleanliness and health.

The initial step in the purchase of any product for the army is taken by the Purchasing Quartermaster. From the approved list of bidders—those business firms which have satisfactorily passed sanitary inspection—he selects the houses dealing in the products involved in a pending contract, and sends them an invitation to bid in which are stated the grade, quality and size, the Government specifications covering each product and the amount desired of each product. The date and place where the



Signal Corps U. S. Army

The army purchases inconceivable numbers of eggs. All are inspected by veterinary officers at the time of purchase and again at issue to troops. Here two students in the School of Meat and Dairy Hygiene, Lieut. Jas. W. Heaton and Capt. Max H. Carlin are receiving instructions in egg candling

bids will be publicly opened and the contract awarded are included. On the date specified representatives of the houses who wish to bid gather at the designated place (usually at the Quartermaster Depot) and the bids are considered. The contract is awarded to the firm which makes the lowest bid. The successful bidder then receives a purchase order which states the time delivery is to be made and, in the case of meat, meat-food or dairy products, that inspection will be conducted under the super-

* Excerpts from an address by Lieut. Col. Fred C. Waters, V.C., Depot Veterinarian, Chicago Quartermaster Depot, at annual meeting of the American Meat Institute.

Signal Corps U. S. Army

The late Col. Will Griffin demonstrates inspection of smoked hams to a class in Meat and Dairy Hygiene

vision of a designated veterinary officer.

After a contract for a meat-food or dairy product is awarded by the Chicago Quartermaster Depot, our office is notified and a copy of the purchase order is supplied. If the product is to be prepared in Chicago our veterinary officers conduct the necessary inspections. Their work begins in the chill room where the meat is offered for sale. The initial inspection, in which the meat is graded to accord with specifications, is the most rigid given, and the most important. Meat passed for processing at that time will probably be finally accepted unless something develops during the processing. Products are house inspected before presented to the Veterinary Corps and if rejected is a loss to the packer.

Lately, the army has been purchasing huge quantities of frozen, boneless beef. For this the carcasses are selected in the chill room by a veterinary officer who places the army stamp of acceptance on those which meet specifications. The beef is graded as low, good, or No. 3, in conformity with rules of the American Meat Institute. Until recently, only steer carcasses were acceptable, but due to the huge quantities which are being purchased heifer carcasses also are being accepted at present. After selection in the chill room, the carcasses are taken into the boning room where they are quartered, cut and boned under the supervision of a veterinary officer and his detail of enlisted men. The meat is then wrapped and packed in a box with the weight and classification indicated and immediately placed in a sharp freezer with the temperature ranging from -17 to -22° F. However, shipments are not permitted to leave the plant until the meat is frozen solid. It is then transported in refrigerator cars. Upon its arrival at one of the refrigerator warehouses, which are located in various sections of the country, it is stored until needed by troops in that section. The use of frozen boneless beef has proved highly satisfactory as it minimizes shipping problems since the product requires much less space than that of other forms of beef.

Each article purchased by the Government is prepared according to specifications compiled by the Government. They are definite, and should be readily understood inasmuch as they are clearly worded; however, at times some difficulty has arisen with various packers regarding the interpretation of the specifications. In one instance a contractor made a bid to supply calf liver under the impression that the product involved was beef liver and he figured on beef liver prices. The Govern-

ment accepted his bid with no knowledge of the fact that the contractor intended to supply beef liver. Of course the meat packer had to deliver the goods in compliance with the contract even though he suffered a tremendous loss. Such gross disregard of contract specifications seldom occurs but when it does it causes a great deal of friction. As a rule the relationship between packer and inspector is a cordial one.

The Chicago Quartermaster Depot awards many contracts which are to be completed outside Chicago. If a contractor needs the services of a veterinary officer, and none is available in the immediate vicinity, he may request that an inspector be sent even from another corps area. The Corps Area Veterinarian assumes the responsibility of assigning an officer for the inspection.

At the direction of the Surgeon General's Office in Washington, our office in Chicago has become a center for supplying these inspectors. In the future, we expect to be sending out more and more officers, particularly on those contracts which are awarded by the Chicago Quartermaster Depot.

A standardized system of inspection throughout the United States is highly desirable. Ill-feeling is caused when one inspector accepts a shipment and the next inspector rejects it on some technicality. Such occurrences cause unfriendliness among the army inspectors as well as between the Government and the packers. Accordingly a School of Meat and Dairy Hygiene for Veterinary Officers was established.

The purpose of this school is to teach all veterinary officers a standard method of inspection which will insure the purchase of good food of uniform quality throughout the army. Such a system of inspection also affords protection to the meat packers by offering assurance that all packing houses are dealt with in the same manner. Each class is made up of approximately 20 officers who come from all parts of the country. The course of instruction is very complete and the school has functioned very satisfactorily.

The meat packers of Chicago have most willingly cooperated with the instructors in connection with the School of Meat and Dairy Hygiene. The officers attending this school have gained a great deal of practical experience by being allowed to use packing plant facilities, and also from the informal lectures delivered by packing house experts.

Signal Corps U. S. Army

CLASS IN THE ARMY SCHOOL OF MEAT AND DAIRY HYGIENE

Left to right: Lieuts. Geo. Snook, J. B. Couch, P. F. Landis, E. J. Sunderville, O. L. Bailey, B. B. Vail, W. S. Bentham, Myron Thom, H. D. Smith, A. W. Winter, Capt. M. H. Carlin, Lieuts. Mawwell, T. M. Eagle, Geo. Walmsley, Capt. V. E. Ishee, Lieuts. J. W. Heaton, J. J. Keane, E. A. Beckcom, Capt's. A. W. Crawford, R. Cook (Instructor) and Lieut. Col. Will C. Griffen (deceased) Director.

Studies and Observations on **the Laboratory Diagnosis of Anthrax**

By C. D. STEIN,[1]
Washington, D. C.

ANTHRAX, from both an economic and public-health standpoint, is probably one of the most serious diseases with which the veterinary profession has to deal. An early positive diagnosis of anthrax is usually helpful in preventing further spread of the disease, while failure to diagnose it may have disastrous consequences. The laboratory examination of specimens for the presence of anthrax on which final and conclusive diagnosis is based, therefore, entails a grave responsibility and, to preclude the possibility of error, should be performed skillfully.

While some veterinarians and laboratory workers consider that anthrax can be diagnosed easily and quickly by microscopic examination, experience has shown that it is hazardous to base a diagnosis entirely upon examination of blood smears. Many organisms, particularly those found in specimens undergoing putrefaction, may be confused morphologically with *B. anthracis* and, even though the microscopic picture appears conclusive, it should always be checked by both cultural and laboratory-animal inoculation tests to guard against possible error in diagnosis. It is a general policy in the Bureau's laboratories to make a positive diagnosis of anthrax only when a tentative diagnosis based on microscopic examination and cultural tests is supplemented by positive animal inoculation tests. By the simultaneous use of these three methods, more than 80 positive cases of anthrax were diagnosed during the last eight years in specimens received for examination.

Importance of Submitting Suitable Material for Examination

In order to insure a quick, conclusive and reliable examination, it is highly essential that suitable specimens be properly collected and prepared for shipment so as to reach the laboratory in satisfactory condition.

Containers in which specimens are shipped should be labelled "Suspected anthrax" and each specimen should be properly identified. A history of the case should be furnished when available.

The importance of collecting specimens from the dead animal soon after death and before putrefaction occurs, as well as using clean, sterile instruments and containers in the process to avoid contamination with extraneous organisms, cannot be overestimated. Ears, spleen tissue, and blood samples when collected in sterile containers from animals a short time after death and brought to the laboratory within a few hours make very satisfactory material for examination, but many of the specimens of this type, particularly those shipped great distances and not properly prepared for shipment, arrive in varying stages of decomposition or may show considerable contamination with organisms other than anthrax.

With fresh specimens laboratory workers, who have had considerable experience with the disease, encounter little or no difficulty in making a tentative diagnosis on preliminary microscopic examination and culture tests, which usually can be confirmed within 48 hours by laboratory-animal inoculation tests. However, when the material submitted for examination has undergone considerable putrefaction, difficulties and delay in diagnosis may arise due to the presence and multiplication of saprophytic organisms resembling anthrax and to a decrease, or even total destruction, of the anthrax organism due to putrefactive changes. Furthermore considerable difficulty may also be encountered in isolating anthrax from material heavily contaminated with extraneous organisms antagonistic to anthrax, such as *B. coli*, *Pseudomonas aeruginosa* (*Ps. pyocyaneus*) and other types of organisms, especially those characterized

[1] Pathological Division, Bureau of Animal Industry, Agricultural Research Administration.

by a rapid spreading growth such as *B. proteus*.

Long experience in laboratory diagnosis of anthrax has demonstrated that the most satisfactory material for examination when shipped great distances consists of blood which has been collected on swabs, blotting paper, or glass slides, and which has been allowed to dry and therefore sporulate. For this purpose blood may be collected from the jugular vein by means of a sterile needle, or from the vessels of the ear or tail, or specimens from the spleen may be obtained through an incision between the ninth and tenth ribs. In carcasses showing evidence of decomposition blood samples should be taken from a peripheral blood vessel.

Since opening an anthrax carcass promotes the formation of anthrax spores, is apt to establish a soil infection and increases greatly the danger of spreading the infection, postmortem examinations, which are extremely hazardous to the operator, should be performed only when necessary. For example, the history connected with at least three recent, suspected cases of anthrax in cattle where a positive diagnosis was made in this laboratory indicates that the infection was transmitted to individuals, who failed to take the necessary precautions to prevent infecting themselves, while performing postmortem examinations.

With the object of determining a satisfactory method of collecting specimens, a series of experiments were carried out on animals dying during an extensive field outbreak of anthrax and on a large number of sheep that died from experimentally induced anthrax. As a result of these studies, it was demonstrated that one of the safest and most satisfactory methods of collecting anthrax specimens, especially for forwarding long distances by mail to the laboratory, consisted in the use of *sterile* cotton swabs attached to a cork that fitted a *sterile* vial (Fig. 1). The properly identified vial containing the swab specimen with the cork securely attached, can be wrapped in paper or cotton to prevent breakage, placed in a double container, preferably small metal mailing tubes, and forwarded to the laboratory for examination. Anthrax organisms could be readily isolated from such swabs saturated with blood or impregnated with material from the spleen of animals dead of anthrax and which had been allowed to sporulate by drying, even after being kept at room temperature for long

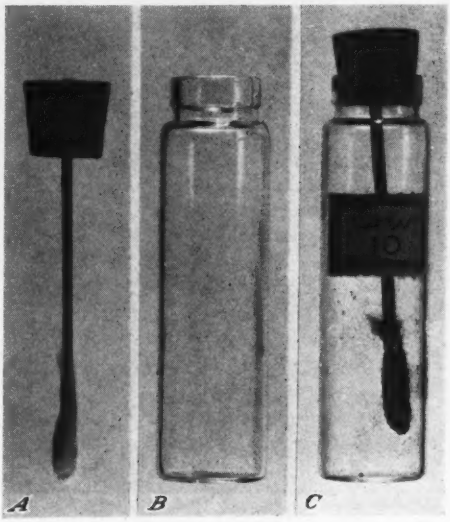


Fig. 1. A. Sterile cotton swab attached to cork. B. Sterile vial. C. Swab specimen in labeled vial ready to be forwarded to the laboratory

periods of time. Virulent anthrax spores were demonstrated readily after more than four years, by cultural and pathogenicity tests of a number of swab specimens prepared from field cases in an anthrax outbreak occurring in South Dakota in 1937. The advantages of using sterile cotton swabs for collecting specimens of suspected anthrax are threefold: (1) safety and cleanliness in collecting specimens, (2) rapid sporulation of the anthrax bacillus with little opportunity for development of putrefactive organisms inimical to anthrax, and (3) the small size of the specimen facilitates handling, packing, and shipping, thus eliminating the hazard of sending large parcels of dangerous infectious material such as blood samples, spleen tissue, or ears through the mails, the fluids of which sometimes leak from containers and soil outside wrappings.

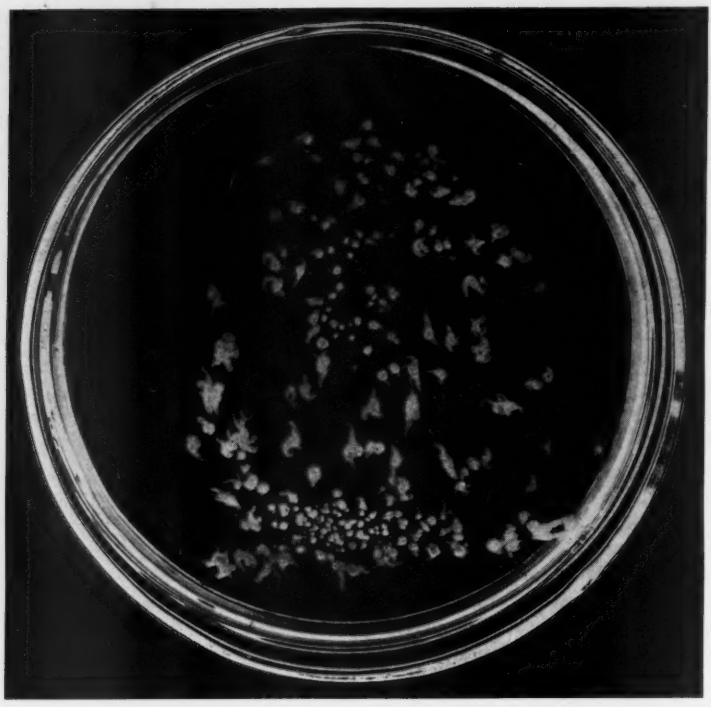


Fig. 2. Anthrax colonies, natural size, on surface of plain agar plate, 24-hour growth, made direct from saline blood suspension from bovine field case. Note irregular shape of colonies, many with tails (comet shaped)

Laboratory Examination

Since the time element is an important factor in anthrax diagnosis, specimens should receive prompt attention. As a preliminary step, smear preparations are first made from the blood, spleen, swabs, or other material submitted. A microscopic examination is immediately made of the smear preparations after fixing, and staining with methylene blue or other suitable stain. The M'Fadyean methylene-blue reaction, which consists in fixing the dry blood films by passing through a flame three or four times and staining with polychrome methylene blue, usually works well on fresh blood specimens. The organisms appear as straight, square-ended, dark blue rods in short chains end to end or often singly or in pairs. Around the organism is noted an amorphous, purplish, stained material which represents the disintegrated capsules (the M'Fadyean reaction). When carefully carried out, the Giemsa or Hasting blood stains are also excellent methods for revealing the capsule (Plate I, Figs. A, B, C, and F).

In microscopic examination of stained smears, prepared from dry material, such as blood or spleen material on swabs, disintegrated organisms, shadow forms, sporulating organisms, or the small, oval-shaped, refractile spores in chain formation, singly or in pairs, may be observed (Plate I, Figs. D, E, G, and H).

If microscopic examination indicates little or no contamination with organisms other than anthrax, saline suspensions are made from the material and a few drops are inoculated on the surface of a series of hardened, plain agar plates, pH 7.2, with a platinum spreader. The plates are incubated at 37.5° C. overnight, or for about 18 hours. One who is familiar with the appearance of anthrax colonies isolated *direct* from blood or spleen material can usually determine by examination with a hand lens whether or not the colonies are character-

istic of anthrax. Such colonies after 18-hours' incubation are small and irregular in shape, many of them showing projections or curved tails (comet shaped. Fig. 2). They are grayish-white, flat, dry, granular, with sort of a ground-glass appearance, especially at the margins, and with a friable or stringy consistency (Plate 2, Figs. B and C). When examined under low magnification, they appear as a mass of closely twisted threads with a dense center and a lighter, wavy margin (Fig. 3 and Plate II, Figs. D and E). Under higher magnification the margin of the colony shows the continuous convoluted parallel arrangement of closely packed, long chans of bacteria, giving the so-called hair lock appearance characteristic of colonies recently isolated direct from fresh blood specimens (Plate III).

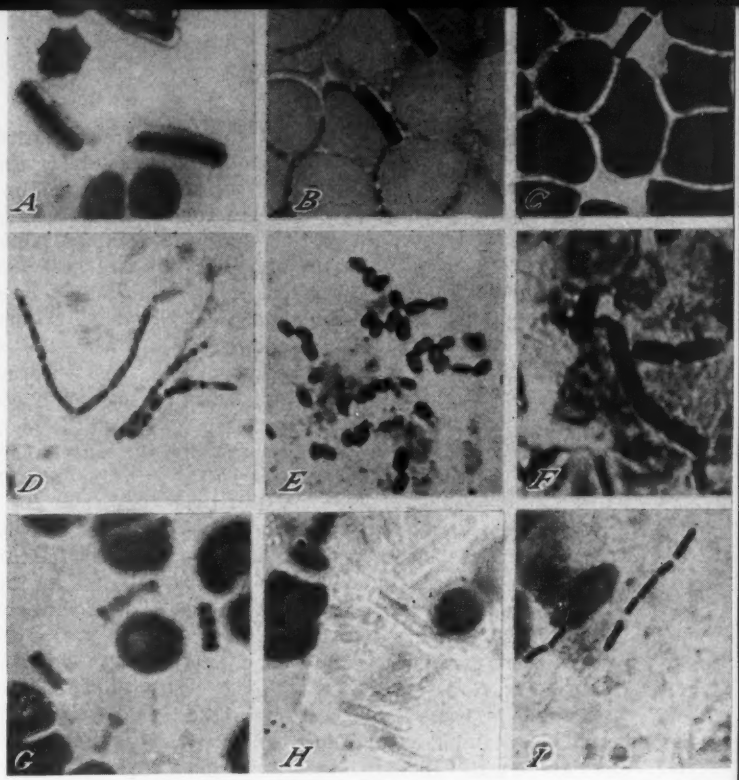


Plate I. STAINED ANTHRAX BACILLI AND SPORES

A. Bacilli, in sheep blood (methylene blue) x 1700. B. Bacilli, guinea pig blood (Giemsa) x 1600. C. Bacilli, guinea pig blood (Hasting's) x 1600. D. Sporulating organisms from spleen (bovine field case) x 1400. E. Anthrax spores from culture x 1900. F. Bacilli in blood, stained with methylene blue (photograph taken with polarized light) x 2000. G-H. So-called shadow or ghost forms x 1500. I. Bacilli, spleen, guinea pig (note long chains) x 1400

Colonies made from virulent broth cultures or from rough-type vaccine cultures have the same general appearance as those made direct from specimens of blood or spleen from field cases, but are more or less circular in shape with wavy irregular margins and no projections. Typical colonies from the agar plates are transferred to bouillon and agar slants for motility, Gram staining, reactions on diagnostic media, and further laboratory animal inoculation tests, if necessary.

In addition to the typical colony formation of virulent field strains of *B. anthracis*, certain other characteristics assist in its identification and differentiation from spore-bearing aerobic organisms resembling it. It is gram positive and nonmotile and in the blood stream appears as a square-ended, encapsulated rod usually in short chains. It is pathogenic for laboratory animals, produces a typical inverted pine tree growth in gelatin slowly liquefying the medium. It produces little or no turbidity in broth cultures and has a weak hemolytic action on blood agar plates, most strains producing little or no hemolysis in 18 hours.

Based on studies of fermentation reactions of 25 virulent strains it produces acid but no gas in dextrose, saccharose, trehalose, maltose, levulose and dextrin. Some strains produce late and slight acidity in glycerol and salicin. No definite fermentation occurs in lactose, galactose, sorbitol, inulin, inositol, adonitol, mannitol, mannose, rhamnose, arabinose, raffinose, and dulcitol.

It slowly acidifies, coagulates, decolorizes,

and finally peptonizes litmus milk. It reduces nitrates, but fails to form indol.

Simultaneously with original seeding of agar plates on the day specimens are received, at least two guinea pigs are inoculated subcutaneously with the saline suspensions made from the specimen. If the material contains anthrax, the guinea pigs usually die in 36 to 72 hours and show characteristic lesions, such as subcutaneous edema in the area of inoculation, dark-colored uncoagulated blood, an enlarged, dark-colored, friable spleen and a congested, mahogany-colored liver. The organism can be recovered readily from the blood of the dead guinea pigs, and microscopic examination of heart blood smears reveals organisms occurring singly or in short chains with the typical morphology of anthrax (Plate I, Figs. B and C). In spleen smears the organisms usually occur in longer chains (Plate I, Fig. I).

However, in those instances where the material containing anthrax is grossly contaminated with anaerobic spore-bearing organisms, the test may miscarry and the guinea pigs may die of anaerobic infection in 24 hours or less without showing any changes characteristic of anthrax. On the other hand, specimens are occasionally encountered where anthrax can be isolated from contaminated material only by animal inoculation tests and where usual culture methods are negative.

Isolation of Anthrax Bacilli from Material Contaminated with Spreaders and Non-spore-bearing Organisms

Hagan[1] in 1920 described a simple method for diagnosis of anthrax in putrefying animal tissues, which was based principally on rapidly distinguishing between colonies of true anthrax and colonies of anthrax-like organisms growing on the surface of agar plates by microscopic examination under high magnification. This method consists in removing the lid of the Petri dish and placing a flamed cover slip over the surface of the suspected colony. The open plate is placed on the microscope stage and the border of the colony is studied with a highpower objective. The morphology of the organism, its arrangement in the colony, spore-formation and, if a small amount of moisture is present on the plate, the degree of motility in young actively motile cultures such as *B. subtilis* can thus be ascertained. This method has frequently been used by the writer and, although somewhat dangerous, is no doubt of considerable assistance in differentiating colonies of field strains of the anthrax bacillus from colonies of spore-bearing aerobic bacilli resembling it, especially when supplemented by pathogenicity tests, but in those instances where the material is contaminated with rapidly growing organisms that crowd out, overgrow, and obscure the anthrax growth on agar plate cultures, it is of limited value.

When the material is contaminated with rapidly growing organisms of the spreader type or with organisms to be discussed later that are especially antagonistic to the growth or pathogenicity of the anthrax bacillus, in many instances *B. anthracis* cannot be recovered by ordinary *cultural* tests or by primary pathogenicity tests.

In some cases anthrax can be recovered from specimens containing extraneous organisms by seeding a few drops of highly diluted suspension of the material over a great surface of plain agar. This is accomplished by seeding a series of plain agar plates (four or five in number) by means of a platinum spreader, the isolated anthrax colonies appearing on the fourth or fifth plate. This method, however, is of little value in dealing with the rapidly growing spreader type of contaminant.

Heating saline suspensions of contaminated material at 65° C. for 60 minutes or at 80° C. for 30 minutes and then seeding is also an effective method of destroying non-spore-forming organisms and of facilitating the isolation of anthrax.

In studies carried out by the writer on this problem, it was found that the treatment of contaminated material with phenol was an excellent method of inhibiting the growth of organisms of the spreader type. In using this method 0.1cc of a saline suspension of the contaminated material is placed in a tube containing 10cc of 1% or 2% phenol, depending upon the degree of contamination. The solution in the tube is thoroughly agitated and, after one hour

at room temperature, a few drops of the phenol suspension is seeded on the surface of plain agar plates. In a few cases, using the same method, the writer has isolated anthrax from heavily, contaminated material by placing 0.1 cc of saline suspension of the material in 10cc of 5% phenol for 30 minutes. This method does not destroy all types of contaminating organisms such as spore-bearing aerobes, but is particularly effective against many of the ordinary concontaminants, especially the spreader type, while anthrax usually resists the treatment. In a number of instances by culturing material so treated it was possible to isolate anthrax in pure culture from contaminated specimens that were previously negative by the usual laboratory procedure. (Plate IV).

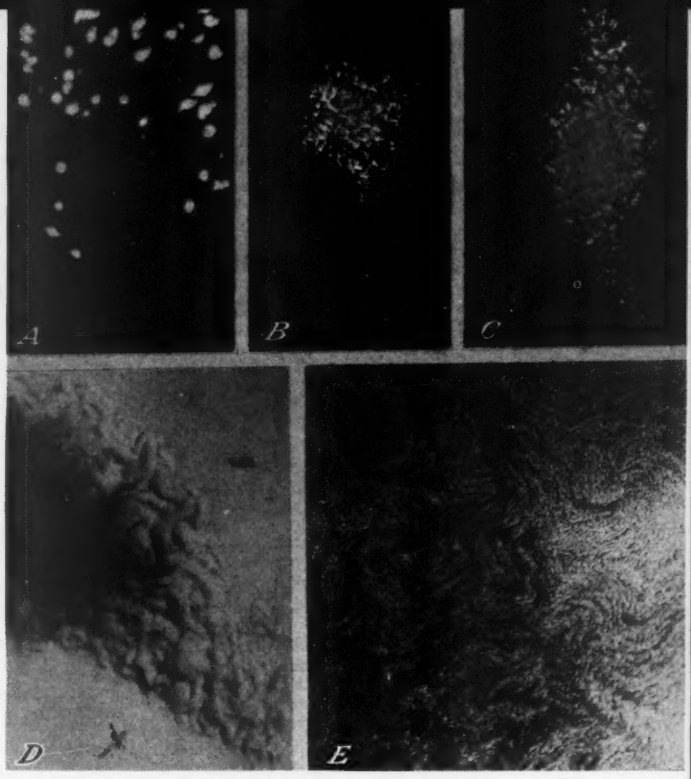


Plate II. ANTHRAX COLONIES ON SURFACE OF PLAIN AGAR PLATES

18-hour growth. Made direct from saline suspension of spleen swab from bovine field case. A. Small and medium, irregular shaped colonies, natural size. B. Small colony, note ground glass appearance x 10. C. Large comet shaped colony, note ground glass appearance x 10. D. Portion of colony (c) showing border x 30. E. Border of colony (c) x 100

A number of investigators have shown that organisms such as *Pseudomonas aeruginosa, Eberthella typhosa, Escherichia coli,* Friedlander's bacillus, and various staphylococci may be antagonistic to the anthrax bacillus, retarding its growth and inhibiti ngor destroying its virulence. In a limited study by the writer on the antagonistic effect of different types of organisms on the pathogenicity of anthrax, using guinea pigs as test animals and the subcutaneous method of inoculation, the following results were obtained:

1. Broth cultures of *B. mycoides* and *B. mesentericus* when mixed with broth cultures of virulent anthrax just prior to injection had no antagonistic effect on the pathogenicity of anthrax, but broth cultures of *B. cereus* and *B. subtilis* when mixed with anthrax cultures and injected under the same conditions had a moderate inhibitory action on the virulence, prolonging the death point from two to three days.

2. In a series of experiments where broth cultures of virulent anthrax were mixed in equal proportion with broth cultures of such soil organisms as *B. mesentericus, B. mycoides, B. flexus, B. megatherium, B. circulans, B. simplex, B. pyogenes, or Erysipelothrix rhusiopathiae* and incubated for 18 hours before injection, little or no inhibitory action occurred in the virulence of the anthrax organism. Cultures of *B. cereus, B. subtilis,* Pasteurellae, streptococci, and staphylococci, when mixed with virulent broth cultures of anthrax and grown and injected under the same conditions had a mild to moderate inhibitory action on the virulence, prolonging the death point from one to three days, while cultures of *Esch. coli* and *Ps. aeruginosa* when mixed with virulent cultures of anthrax and grown and injected under similar conditions had a marked antagonistic effect, prolonging the death point from two to five days, and in the case of *Ps. aeruginosa* in some instances entirely destroying the virulence.

In connection with these studies it was demonstrated that broth cultures of each of the following, *B. cereus, B. siamensis, B. subtilis, B. mycoides, B. mesentericus, B. anthracis (avirulent),* and a number of unidentified spore-bearing soil organisms,

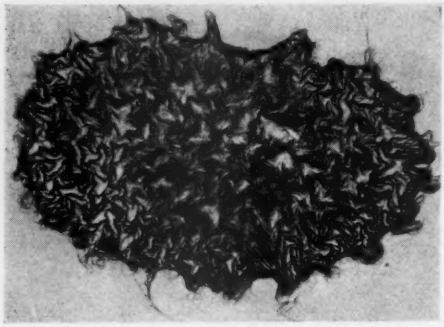


Fig. 3. Anthrax colony (x 30) removed from agar plate, fixed, mounted, and stained lightly with Giemsa to show arrangement of bacterial filaments throughout colony. Loose filaments at margin are due to manipulation in fixing and mounting

when injected in 1 to 2cc doses, subcutaneously, failed to immunize guinea pigs that were subsequently exposed to virulent cultures of *B. anthracis.*

It was also shown that laboratory cultures of *B. anthracis* carried on plain agar slants for at least 11 years, when transferred every 30 to 60 days and held at a temperature of 3 to 5° C., remained fully virulent for guinea pigs, producing death in 48 hours or less.

Diagnosis of Anthrax in Swine

Anthrax in swine is usually the result of feeding on anthrax carcasses or other infected material. The disease in hogs is usually accompanied with an extensive edema of the throat, sometimes referred to as cervical or gloss anthrax. Swine are very resistant to anthrax and, while a number of hogs may die with the acute septicemic anthrax following an outbreak in a herd, a considerable percentage will develop the disease in the chronic form or make a gradual recovery. However, when some of these animals are presented for slaughter, evidence of anthrax infection may be found in the cervical lymph glands, which in the early stages of the disease are usually enlarged, hyperemic, and brick-red in color, and, if the tonsils are involved, they may show areas of necrosis with edema of the adjacent tissue. As a rule anthrax can be recovered from such lesions, but in the more chronic form in which there is marked fibrosis with other evidence of healing, there may be difficulty in recovering the organisms. A microscopic examination of smears prepared from such lymph glands may in some cases reveal only disintegrated or shadow (ghost) forms of the organism. In some cases it may be possible to cultivate these organisms, but they may not be pathogenic for laboratory animals.

While spleen specimens from hogs dying of acute anthrax are occasionally received for examination in this laboratory, most of the specimens received consist of lymph glands from suspected cases of chronic cervical anthrax retained on postmortem examination in establishments operating under Federal meat inspection. In handling specimens of this kind it has been our experience that glands showing lesions suggestive of anthrax infection in which anthrax organisms could be demonstrated microscopically on preliminary examination may on further study result in any one of the following findings:

1. Positive, both culturally and on animal inoculation (guinea pigs and mice).
2. Positive culturally and pathogenic for mice but not for guinea pigs.
3. Positive culturally and non-pathogenic for both guinea pigs and mice.
4. Negative, both culturally and on laboratory animal inoculation.
5. Positive culturally from some sections of a given gland and negative from other sections of the same gland.

Doctor Stiles, in charge of the Bureau's branch pathological laboratory in Denver, Colo., reports similar results in handling specimens of this kind. Due to the fact that only a limited amount of material can be examined under laboratory conditions, hogs which on post-mortem examination show evidence of anthrax infection on gross appearance and on laboratory examination reveal only microscopic evidence of the presence of the organism, are considered

as infected with anthrax and disposed of on that basis.

Recovery of Anthrax from Miscellaneous Specimens of Unusual Interest

Recovery of anthrax from the blood of a cow 48 hours before death.—During an outbreak of anthrax in North Carolina in 1941, a blood sample collected from the jugular vein of a sick cow was forwarded to this laboratory for examination. The animal was visibly sick about 12 hours previous to the time the blood sample was collected, had a temperature of 107° F., and died 48 hours subsequent to the collection of the sample. Anthrax was isolated from this specimen by laboratory-animal inoculation tests: Guinea pigs inoculated subcutaneously with a saline suspension prepared from the sample died in from 48 to 72 hours from anthrax. In cases of this kind, when an anthrax bacteremia exists several hours prior to death, the infection may be mechanically transmitted by blood-sucking flies from infected to healthy animals. In this connection it is interesting to note that Barron[2] in 1940 also reported a case of early septicemia in a case of anthrax in which the bacilli were present in the blood stream at least 30 hours before death; while Kraneveld and Djaenoedin[3] in 1940 microscopically demonstrated the presence of anthrax organisms in the blood of a buffalo 10 hours before death and in the blood of horses 2 to 16 hours before death. The same authors transmitted the disease from infected horses and buffaloes to healthy animals with *Tabanus rubidus* Wied and concluded that tabanids played a considerable part of the spread of anthrax.

Isolation of Vaccine Anthrax from the muscles of a beef carcass.—In one of three cases examined, *B. anthracis*, with the cultural characteristics and virulence of a No. 2 Pasteur vaccine strain, was isolated from a muscle specimen from a beef animal. The muscle specimen, which showed inflammation and tumefaction, was taken from the usual site of anthrax vaccination in a steer, the carcass of which had been retained as a suspect at an establishment operating under Federal meat inspection. This finding has a practical bearing in that it indicates attenuated anthrax spores may persist for a considerable period at the site of inoculation following vaccination. It demonstrates the advisability of holding animals used for food purposes for several weeks following treatment with anthrax spores, before slaughtering them for food.

Federal meat inspection regulation issued in Circular Letter No. 2276, Oct. 17, 1940, was promulgated to meet this contingency.

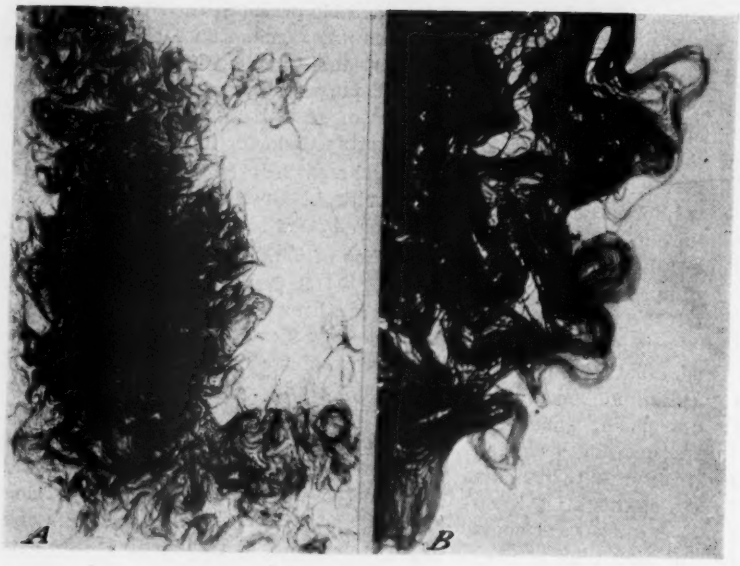
Isolation of anthrax from a dog and from flies.—Dr. M. S. Shahan, of this laboratory, isolated anthrax from specimens of house flies and blow flies caught while feeding on the carcass of a steer dead of anthrax in an outbreak that occurred in South Dakota in 1932. This finding indicates that the non-blood-sucking flies also may play a role in the dissemination of anthrax spores. He also isolated anthrax from a section of dog spleen in 1933. The specimen was from one of three dogs that died suddenly subsequent to eating meat from a cow that had died under mysterious circumstances.

A point of considerable interest in connection with the spread of anthrax by flies is that the infection may be transmitted to man by the bites of flies. Two such cases were reported to the Bureau in recent years, one during an outbreak in Texas and another, which proved fatal, during an outbreak in South Dakota.

Since reports indicate that some of the most severe and rapidly spreading outbreaks of anthrax occur at the height of the fly season and quickly abate with appearance of cold weather and disappearance of flies, it would appear that flies play a greater role in the spread of the infection than is generally supposed. Fly control, therefore, is a factor that should receive more attention in measures for the control of this disease.

Effect of Freezing on the Viability of *B. anthracis*

In tests to determine the effect of freezing on the viability of *B. anthracis*, a culture of anthrax in serum broth made in February, 1934, quickly frozen and held continuously at a temperature of —5 to —10° C. until February 1942 (a period of 8 years), was found to be both viable and virulent



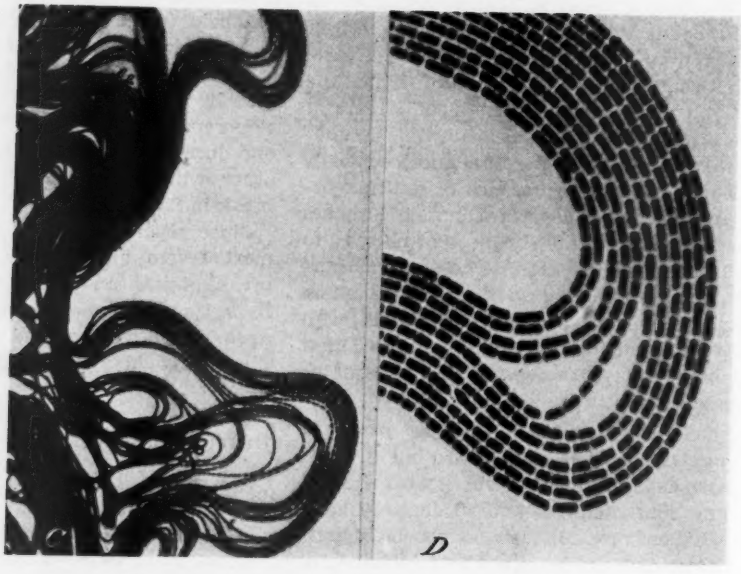


Plate III. PHOTOMICROGRAPHS OF AN ANTHRAX COLONY

Removed from surface of plain agar plates by impression method, fixed, mounted, and stained with methylene blue. A. Colony from anthrax field strain (x 45). B. Border of same colony (x 145). C. Extreme margin of a portion of the border shown in B. (Note the wavy hairlike, parallel arrangement of bacterial filaments composing upper and lower loops (x 400). D. Upper loop shown in C highly magnified to show arrangement of individual bacteria composing the long chains (x 1600)

when examined by cultural and laboratory-animal inoculation tests.

Summary

It is necessary to make a thorough laboratory examination in all cases of suspected anthrax and it is advisable to confirm such tentative diagnoses based on microscopic examination and culture tests, by animal-inoculation tests.

It is important to submit only suitable material for laboratory examination. The use of sterile cotton swabs for the collection of specimens in the field is the most convenient method.

A practical procedure for carrying out laboratory examination for anthrax, the typical colony formation on plain agar plates of cultures made direct from blood or spleen specimens, and certain other characteristics that assist in the identification of *B. anthracis* and its differentiation from anthrax-like organisms are described.

The use of phenol to facilitate the recovery of anthrax from material contaminated with non-spore-bearing organisms, especially those of the spreader type, is the method of choice.

Possible laboratory findings in chronic cervical anthrax in swine and the antagonistic influence of a number of different organisms on the pathogenicity of anthrax that may complicate the diagnosis are given.

A case of anthrax bacteremia was demonstrated in a cow 48 hours before death, thus showing the possibility of spread of the disease by blood-sucking insects. Virulent anthrax organisms were recovered from house flies and blow flies, and from the spleen of a dog that had fed upon the carcasses of animals dead of anthrax. The organisms were also isolated from muscle tissues of the carcass of a beef animal that had been vaccinated some time before slaughter.

There is good evidence that indicates that a culture of anthrax held in a frozen state (—5 to —10° C.) for a period of eight years and that anthrax spores contained in dried blood and spleen material on cotton swab specimens collected from field cases and held at room temperature for over four years were both viable and virulent.

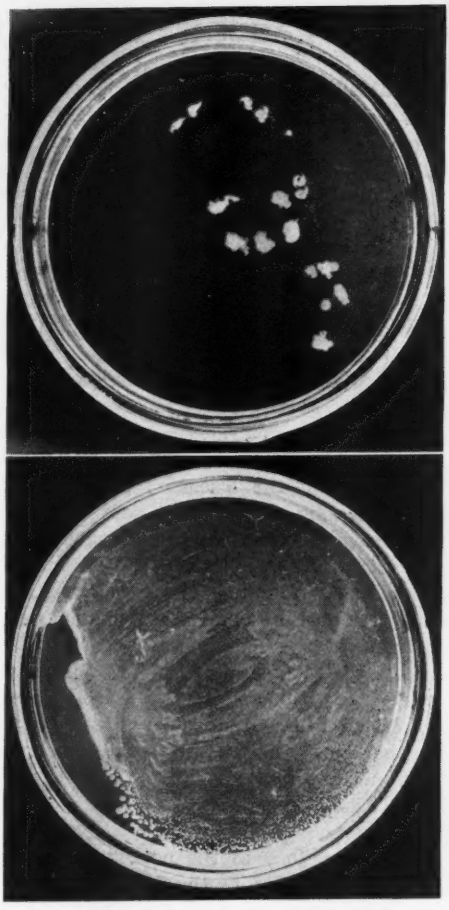


Fig. 4. (Reduced slightly more than one-third) Above: Growth on surface of plain agar plate seeded with a suspension from an anthrax swab specimen contaminated with extraneous organisms. Below: Growth resulting from same suspension after treatment with 2 percent phenol. Note isolated anthrax colonies

Acknowledgement

The writer is indebted to Dr. H. R. Seibold, Pathological Division, Bureau of Animal Industry, for valuable assistance rendered in mounting, fixing, and staining anthrax colonies.

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Authoritative Statements Concerning Trichinosis

RECENT discussions of trichinosis by public health officers and others contain a hint that the time given the packing industry, the swine industry and the state livestock disease control officials to solve the trichinosis problem is running out. In a conference in Omaha in 1937, the late Maurice C. Hall, representing the Surgeon General of the U. S. Public Health Service informed the interests mentioned that trichinosis in man is regarded as a public health problem of sufficient importance to justify a control program; that solution of the problem is not regarded as impossible or particularly difficult, and that practical eradication of the disease from swine or the processing of pork and pork products to destroy the trichinella is regarded as a necessary first step in the suppression of the disease in man. It was pointed out that trichinosis in swine is, in large measure, a disease of hogs fed upon raw garbage and finally that, since only 6% of the hogs marketed in this country are garbage fed, the interest of 94% of the swine industry has been injured by this small fraction. Trichinosis is not only an obstacle to the wider use of pork but likely at any time to bring about boycotts of pork by considerable sections of the population. The hope was expressed by the Surgeon General that the swine industry, the packers and livestock sanitary officials might undertake the task cooperatively to the end that infected pork be eliminated from the market. The belief was held that this could be accomplished by the interests involved at small cost and little injury or inconvenience to anyone, while, on the other hand, if such action must be forced on the swine packing industries by an outside agency the cost might be high, particularly in lost markets. According to Doctor Hall:

"Prompt action looking toward the control of trichinosis is imperative for the protection of public health on the one hand, and the protection and improvement of both the swine and the packing industries on the other hand. An extension of the present move by scientists, physicians, and veterinarians to protect the public health must be anticipated by the swine growers and packares to protect their industries from the consequences which may be expected from research within the next few years.

"Since the control of human trichinosis depends on the control of porcine trichinosis (an interstate problem) it is essential that there be a national control program for the United States, in which the U. S. Public Health Service, as the federal agency concerned with human trichinosis, and the Federal Bureau of Animal Industry, concerned with porcine trichinosis, could cooperate jointly in assisting with coordination of the work of State health departments and State livestock sanitary boards, respectively.

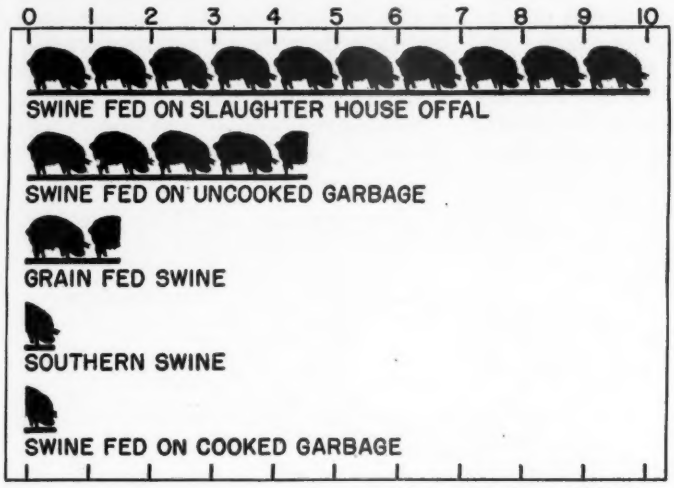
"In this connection some system of licensing may be found desirable in order that more adequate control may be exercised over plants feeding cooked garbage."*

Surgeon General Thomas Parran has stated that one in every six human specimens examined in the laboratory (U. S. Public Health Service) over a period of four years have been found to contain lesions of trichinosis. Applied to the population at large this would indicate that 21,000,000 Americans are or have been infected with this parasite in that time.

Little has been accomplished in the control of swine trichinosis in the five years that have elapsed since the U. S. Public Health Service issued its warning on the subject. A few of the large packers have discontinued the purchase of hogs fed raw garbage, others purchase them only at a discount of 50 cents to $1.00 a hundred from the market price. The effect has been simply to drive such hogs in greater numbers to the small packers who operate without

*Reprint No. 1975, Studies on Trichinosis VII by *Public Health Reports*, Aug. 19, 1938, pp. 2, 11 and 12.

any inspection whatever; thus it makes matters worse. Livestock sanitary officials who really hold a key position in any co-operative control measures to date have given the program little or no support.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SWINE FED ON SLAUGHTER HOUSE OFFAL
SWINE FED ON UNCOOKED GARBAGE
GRAIN FED SWINE
SOUTHERN SWINE
SWINE FED ON COOKED GARBAGE

The type of feed consumed by swine has a direct bearing on the number of cases of trichinosis in humans, as can be seen by accompanying illustration

Courtesy, National Institute of Health

Since the question threatens to become a hotly contested one, if not soon, at latest when the present meat shortage has passed, the following quotations are given that the opinions of experts may be known. It is believed only the most presumptive will question them. At the close of each quotation the page of the publication from which it is taken is noted.

Extent of Garbage Feeding of Hogs, by U. S. Bureau of Agricultural Economics—June 1940.

1. "Over one million hogs annually are being garbage fed and later marketed in the United States." Page 1.

2. "Most garbage feeders do not pay for garbage." Page 2.

3. "47% of these hogs are being fed exclusively on garbage (no grain)." Page 1.

1942 Agricultural Yearbook—Keeping Livestock Healthy.

4. "Hogs originating on the Atlantic and Pacific seaboards and known to have been fed almost exclusively on untreated garbage, 6.11% were found to be infected (trichinous)." Page 797.

5. "Not only was the incidence of infection (trichinae) in the garbage-fed hogs greater than in the farm-raised hogs fed forage, grain and other feeds, but the intensity of the infection in the garbage-fed series was also much greater." Page 797.

6. "Investigations conducted in the United States during the last 12 years and still in progress have disclosed a surprisingly high incidence of infection with trichinae in the general population of this country." Page 794.

7. "Hogs known to have been fed cooked garbage only. Just .5% were found to be infected, the numbers of trichinae in the positive hogs being very small." Page 797.

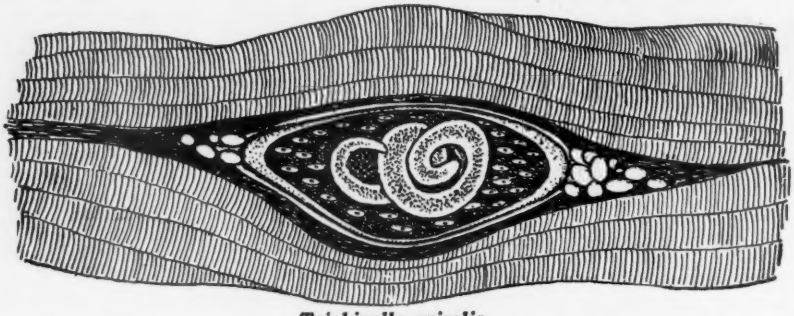
8. "In Canada, where swine-husbandry practices are more nearly similar to those prevailing in the United States . . . an examination discloses the incidence (trichinosis) from garbage-fed hogs to be .2%. In a previous investigation the incidence of the infection was .57%. These low figures are not surprising since Dominion laws require the sterilization by heat of all garbage fed to swine, which is very close to the figure obtained by examinations in the United States of diaphragms of swine fed cooked garbage." Pages 797-798.

9. "In the United States, where uncooked garbage is fed to hogs, the incidence of nonclinical human trichinosis is about 16%." Page 794.

10. "In England, where only cooked garbage is fed to hogs, the incidence of nonclinical human trichinosis is about 1%." Page 795.

11. "In Canada, where only cooked garbage is fed to hogs, the incidence of nonclinical human trichinosis is about 1.6%." Page 795.

Studies on Trichinosis. VII. The Past and Present of Trichinosis in the United States, and the Indicated Control Measures, by Maurice C. Hall, Late Chief, Division of Zoology, National Institute of Health, U. S. Public Health Service. Reprint No. 1975, *from the Public Health Reports Vol.* 53, *No.* 33, *August* 19, 1938. *pp* 1472-1486.



Trichinella spiralis.
Larva encysted in muscle. Enlarged, From Ransom, after Leuckart

12. "Human trichinosis rests primarily on a basis of swine trichinosis. . . . The indicated control of trichinosis in the United States is primarily a matter of keeping such uncooked pork scraps out of the feed of swine, and either cooking such feed as garbage or refraining from feeding any food containing pork scraps." Page 1.

"Potentially, trichinosis could be practically eradicated within 5 years, and this could be done with very little expense." Page 12.

"If the packing plants operating under Federal inspection were to adopt the practice of processing the carcasses of pork fed on garbage, to destroy any trichinae present, and make legitimate deductions for added costs from the price of such hogs, the immediate effect would be to drive such animals to the smaller packing plants and slaughtering establishments not operating under Federal inspection. In general, these plants either have no inspection or have a highly inadequate inspection by non-professional, untrained inspectors. Steps should be taken through the joint efforts of health departments and livestock sanitary boards to establish nation-wide meat inspection by professional, trained personnel along lines equivalent to the standard maintained by the Federal Bureau of Animal Industry. This development is long overdue." Page 13. (*Compiler's comment*: In case states should enact laws empowering state livestock sanitary authorities to impose a quarantine on all hogs fed upon uncooked garbage, then, by the same authority and in the interest of the public health, states could also designate that the hogs in question be slaughtered at a plant where the inspection meets the approval of the state. All such hogs moving interstate would move on a Federal permit. There may be laws now in existence which empower livestock sanitary authorities to impose quarantine on hogs that have been fed upon uncooked garbage. However, because their predecessors in office have not enforced such laws, there is little reason to believe present incumbents will. Therefore, specific new legislation on garbage feeding of hogs is desirable if the interests of public health are to be accorded proper consideration.

Studies on Trichinosis. XIV. A Survey of Municipal Garbage Disposal Methods as Related to the Spread of Trichinosis, by

Willard H. Wright, Reprint No. 2172 from the *U. S. Public Health Reports*, Vol. 55, No. 24, June 14, 1940. Pages 1069-1077.

13. "We have been informed that many garbage feeders avoid marketing their hogs in federally inspected packing plants in order to obviate price differentials resulting from condemnations under Federal inspection for disease conditions, other than trichinosis, which are common in garbage-fed hogs. In uninspected plants, trichinous pork may go into products customarily eaten raw by the consumer, thus providing very dangerous avenues of infection." Page 5.

As of January, 1940, the U. S. Department of Agriculture estimated that at least 600,000 of the 12,250,000 garbage-fed in thlis country were infected with trichinae. If each pig furnishes about 100 meals the infected hogs would provide 60,000,000 trichinae infected meals.

Trichinosis—a Public Health Problem, by Thomas M. W. Cameron, Director, *Institute of Parasitology; Professor of Parasitology, McGill University, Macdonald College*, P. Q., Canada. Reprint from the *Veterinary Journal*, Vol. 93, No. 12, Pages 39-41.

14. "Canada insists on all garbage being cooked before feeding to pigs, although no such law is in force in the United States. This law, at least so far as the larger producers are concerned, seems to be quite generally observed." Page 2.

Studies on Trichinosis. VI. Epidemiological Aspects of Trichinosis in the United States as Indicated by an Examination of 1,000 *Diaphragms for Trichinae*, by the late Maurice C. Hall, *Professor of Zoology, Division of Zoology, National Health Institute, U. S. Public Health Service*, Reprint No. 1954 from the *Public Health Reports*. Vol. 53, No. 26, July 1, 1938. Pages 1086-1105.

15. "Since the great majority of swine are free from trichinae, it seems reasonable that the bad practice of feeding raw and undercooked pork scraps to a small minority of swine should be abolished in the interest of public health. This prohibition of feeding of raw pork scraps to swine has been recommended by previous writers, including Ransom, but adequate action on this vital point has not yet been taken." Page 18.

Trichinosis, by T. W. M. Cameron, Director, *Institute of Parasitology, McGill University, Macdonald College*, P. Q., Canada. Reprinted from *Canadian Journal of Comparative Medicine*, November, 1937.

16. "Human trichinosis is generally subclinical and most cases do not receive medical attention. Clinical cases are by no means infrequent but owing to the great diversity of symptoms, are generally wrongly diagnosed—not even syphilis can imitate so many diseases. These clinical cases are almost always traceable to locally slaughtered pork which is eaten uncooked; in this way quite extensive outbreaks may be caused by a single hog. . . . If the particular pig was infected, the consumer was exposed to clinical trichinosis because he swallowed a relatively large number of cysts. This is still the case with the private producer but it has ceased to be the practice with the large manufacturers. They use mixed trimmings from many hogs, only a few of which are infected, so their sausage contains only a few cysts at most, and resulting clinical trichinosis is unlikely."

The Veterinary Director General of Canada, A. E. Cameron, says that cooking of garbage is not expensive and many feeders believe that hogs thrive better on cooked garbage. Canada requires that all such feed be cooked. A resident of Canada is one-twelfth as likely to become infected with trichinae as is one who lives in the United States where uncooked garbage is fed to hogs.

Further Studies with Sulfaguanidine in the Control of Ovine Coccidiosis

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SINCE the introduction of sulfaguanidine as a medicament for the treatment of bacterial infections of the intestine[6] several investigators have tested the value of this compound for controlling coccidial infections of domestic animals and poultry. The use of sulfaguanidine in experimental poultry coccidiosis[1, 3, 5] suggests in general that this drug affords good protection against the several species of coccidia that cause severe disease but that it is not useful as a curative agent. Chicks fed mashes that contained 1% or more of the drug were found to gain weight somewhat more slowly than uninfected control birds, but the findings as to the efficacy and safety were sufficiently encouraging to augur the possibility of successful chemical prophylaxis against this important disease. In limited studies on experimentally infected calves, daily doses of 0.1gm per kilogram of body weight were found[2] to afford adequate protection against coccidiosis, provided the administration of the drug was begun before or soon after artificial inoculation. These studies also suggested that daily doses of about 5gm per calf, if started with the first appearance of the symptoms (bloody diarrhea and high oocyst output), may cure the disease. Similarly encouraging results were obtained in studies on naturally acquired coccidial infections of sheep[4]. Under conditions of moderate exposure to infection, daily doses of 1gm in lambs gave adequate protection, while doses of 3gm daily, given after the development of high oocyst production, seemed to interfere with production of normal oocysts. On the other hand, available information suggests that sulfaguanidine may be of little or no value in the treatment of canine coccidiosis[7]. The course of the infections in naturally infected puppies was not altered by administrations of approximately one grain of the drug per pound of weight, in three divided doses daily for eight days. It is evident from these studies that sulfaguanidine has promise of being a safe and effective agent for preventing coccidiosis among some of the domestic animals and poultry, and that it may be useful, in some cases, as a cure.

The present report relates the results of a small field test conducted in central Nebraska, in which sulfaguanidine was used against experimentally induced coccidiosis in feeder lambs.

Procedure

The lambs used in this experiment were part of a shipment from Texas that had been four days en route to the feedlots. Immediately after arrival, twelve experimental lambs were selected at random and divided into three groups of four lambs each. Each group was confined in a separate small pen, equipped with troughs for feeding and salting, and a tub for watering. The ration consisted of a mixture of chopped alfalfa and ground corn held together with a small amount of molasses and some water. This mixture was kept before the lambs at all times throughout the experiment. Feed troughs were cleaned every other day during the first week to minimize the ingestion of coccidial oocysts reaching the feed through natural contamination.

The infective oocysts fed to each group were obtained from feces, collected during the preceding months, from lambs with clinical coccidiosis. They were sporulated in approximately 2% potassium dichromate solution during the week prior to the infection of the lambs. To remove most of the chemical before infecting the lambs,

*Resigned May 30, 1942.

the oocyst-containing fecal sediment was washed several times with water.

On the day of confinement in the experimental pens, each group was fed enough of this washed fecal sediment to give each lamb approximately 500,000 sporulated oocysts. No additional feed was given until the following morning, when the infective mixtures had been completely consumed. Oocyst intake was, therefore, approximately the same for each group and each lamb. Beginning 24 hours after the infective feed was made available, and for 20 consecutive days thereafter, one group of lambs was fed exclusively a ration containing 0.2% by weight of sulfaguanidine. On the assumption that each lamb consumed on the average about 1,500gm of feed per day, the medicated mixture thus made available to each lamb contained approximately 3gm of the drug daily. A second group of lambs was fed a medicated mixture containing 0.45% sulfaguanidine beginning on the first day that a majority of the animals showed clinical coccidiosis, and this mixture was fed eight consecutive days through the peak of the coccidial infection. It was not possible to determine the average daily consumption of the drug by the lambs of this group because the loss of appetite associated with severe clinical coccidiosis resulted in subnormal food intake. However, small amounts of the mixture were taken by the sick lambs during the first day of treatment, and thereafter consumption increased gradually throughout the remainder of the period. A control group of lambs similarly infected received no sulfaguanidine throughout the experiment.

Frequent oocyst counts were made on pooled fecal samples from each group to determine the magnitude of coccidial infection, and symptoms were noted daily.

Results

For 11 days after experimental infection, the untreated control lambs remained normal. On the 12th day all lambs in this group showed a loss of appetite and two animals had diarrhea. During the eight days from the 12th to the 19th day of the infection period all the lambs underwent severe clinical coccidiosis, the symptoms reaching a peak on the 15th day, when the animals showed extreme emaciation, sluggishness, inappetence, and diarrhea. Two lambs discharged considerable amounts of blood in the feces, one of them dying on the 16th day after infection. Perceptible improvement in the remaining lambs was first noted on the 18th day and diarrhea ceased completely during the next two days. One lamb was still badly emaciated at the 22nd day. Dilution counts showed that the total average oocyst discharge for the group had risen sharply by the 14th day and remained at relatively high levels throughout the rest of the clinical period (Table I, untreated control group).

The group of lambs given a feed mixture containing 0.45% sulfaguanidine as a therapeutic agent for eight days, commenc-

ing on the day after the first appearance of clinical symptoms, roughly paralleled the untreated control group in incidence, severity and duration of symptoms. Two lambs died of coccidiosis, one at the height of the infection, the other after several days of extreme emaciation following acute infection. The total average oocyst count, however, while somewhat higher at its peak than in the control group, diminished progressively following the 16th day of experimental infection, until oocysts could be detected only by flotation during the last week of the experiment. Over one-half of the oocysts recorded in dilution counts, after sulfaguanidine had been incorporated in the feed for two days, were abnormally small, densely granular, and apparently non-viable (Table I, sulfaguanidine therapeutic group).

In the group of lambs given a 0.2% sulfaguanidine feed, one of the four lambs showed a mild, intermittent, non-bloody diarrhea from the 12th to the 21st day of the infection, but failed to develop marked emaciation or loss of appetite. The other lambs of the group remained visibly normal throughout the 27 days of the experiment. Average oocyst production by this group remained extremely low. In pooled fecal samples, dilution count invariably revealed less than 5,000 oocysts per cc of feces, and usually oocysts were detected only by flotation (Table I, sulfaguanidine prophylactic group).

TABLE 1.—EFFECT OF SULFAGUANIDINE ON CLINICAL SYMPTOMS AND OOCYST PRODUCTION IN FEEDER LAMBS INFECTED EXPERIMENTALLY WITH LARGE DOSAGES OF SPORULATED OOCYSTS

	Untreated control group (4 lambs)		Sulfaguanidine therapeutic group (4 lambs)			Sulfaguanidine prophylactic group (4 lambs)		
Date (1941)	Lambs with coccidial dysentery (number)	Total average oocyst count (per cc of pooled feces)	Sulfaguanidine	Lambs with coccidial dysentery (number)	Total average oocyst count (per cc of pooled feces)	Sulfaguanidine	Lambs with coccidial dysentery (number)	Total average oocyst count (per cc of pooled feces)
Nov. 25*								
" 26						p		
" 27						p		
" 28						p		
" 29						p		
" 30						p		+
Dec. 1						p		
" 2					+	p		+
" 3						p		
" 4		+			+	p		+
" 5						p		
" 6						p		
" 7	2	+		1	5,000	p	1	+
" 8	4		t	3		p	1	
" 9	4	67,000	t	4	137,000	p	1	+
" 10	4		t	4		p	1	
" 11	3 (d)	52,000	t	4	220,000	p		+
" 12	3		t	4		p		
" 13	2	27,000	t	2 (d)	57,000	p		+
" 14	1		t	1		p	1	
" 15		30,000	t		10,000	p		+
" 16							1	
" 17		10,000			+			+
" 18								
" 19		7,500		(d)	+			+
" 20								
" 21					+			+
" 22		10,000						

*Each lamb infected with approximately 500,000 sporulated oocysts
(d) Death from coccidiosis
+ Indicates less than 5,000 oocysts per cc of pooled feces
t indicates sulfaguanidine therapy (0.45% in feed)
p indicates sulfaguanidine prophylaxis (0.2% in feed, or about three grams per lamb daily)

Discussion

Of the many substances that have been tested as medicaments for coccidiosis, only elemental sulphur and some of the sulfonamides have shown effective action against the parasites causing this disease. The rela-

tive advantages of these drugs in the control of coccidial infections of poultry were summarized by Levine.[5] In the cases of sheep and cattle, as well as poultry, it appears that sulfaguanidine is the safest and most effective of the drugs that have been tested up to the present time. The relative superiority of this drug is probably due in large measure to its greater water solubility and its low rate of absorption from the gastrointestinal tract.

The results of the experiment recorded in this paper indicate that coccidiosis of feeder lambs can be prevented by the use of sulfaguanidine. The relatively small doses of the drug administered prophylactically to one group of lambs during the incubation and expected clinical periods of the infection effectively prevented the onset of clinical symptoms of coccidiosis and virtually inhibited the production of oocysts. One lamb of this group showed mild scouring but on the basis of previous experience with sulfaguanidine in sheep, it is doubtful that this symptom was due to the drug. The drug showed no value in the group on which it was tested for curative action, but the decline in oocyst production during treatment with sulfaguanidine and the appearance at this time of a high proportion of abnormal occysts suggest the possibility of therapeutic value in clinical infections of longer duration than reported.

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Sulfaguanidine for Cecal Coccidiosis

Sulfaguanidine is one of the newer sulfa compounds. Since it is very insoluble when given by mouth and reaches the intestines unchanged it is absorbed slowly and can act directly upon coccidia or other disease-producing organisms in the intestines. Chickens were found to be so tolerant to the drug that large doses in capsules or mixed with mash could be given without causing definite symptoms of toxicity or death. However, by feeding mash containing sulfaguanidine to growing chickens continuously over 10-day periods, a toxic effect could be shown and measured by comparing the gains in weight of the chickens with those of control chickens fed nonmedicated mash.

For moderate infection, 0.5, 1, 2 and 4% sulfaguanidine mash feeding was started when the chicks were inoculated. The 0.5% sulfaguanidine mash group showed mild symptoms of coccidiosis, the other treated groups none at all. The highest effectiveness was seen when treatment was started a day before inoculation. When feeding of the medicated mash was delayed until two to six days after the birds were inoculated, it was much less effective, although all treated groups fared better than nontreated, inoculated birds.

The experimental results indicate that sulfaguanidine may be of practical use in the control of outbreaks of cecal coccidiosis on poultry farms. To determine this, it will be necessary to conduct controlled trials on infected farm flocks. Experiments to determine if the drug may be of value in the treatment of intestinal coccidiosis are in progress. Sulfaguanidine has not yet been released for general veterinary use and is at present quite costly. Should the future demand require large-scale production, however, it is likely that the cost would be greatly reduced.—*Beach, J. R., Univ. of Calif.*

★ ★ ★ BUY WAR SAVINGS STAMPS AND BONDS

Histomoniasis in Broilers**

By D. F. EVELTH*
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THE protozoan, *Histomonas meleagridis*, is generally considered as a pathogen of primary importance to turkeys. However, Tyzzer[1] and Delaplane[2] both reported that chicks could be experimentally infected with this organism. Van Es and Olney[3] in 1934, suggested that blackhead may become a disease of economic importance in poultry.

From time to time, chicks from four to eight weeks of age have been submitted to this laboratory and found to be heavily infested with organisms morphologically similar to Histomonas. In these cases the ceca, and frequently the liver also, have presented lesions similar to those described for turkeys infested with histomoniasis.

The frequency with which coccidiosis occurs in chicks makes it difficult to make a satisfactory estimate of death losses due entirely to blackhead. However, in one outbreak, in which no evidence of coccidiosis could be found, the owner reported a loss of 250 of a flock of 500 chicks with more showing symptoms but recovering. This outbreak appeared to be one of uncomplicated histomoniasis.

A few transmission experiments have been conducted in order to obtain more information regarding the pathogenicity of this organism for poultry. A 4-week-old chick, showing symptoms and passing the histomonas organism in the feces, was placed in a cage with a 14-week-old chicken. Within a week the older chicken was depressed, had a diarrhea, and numerous histomonas organisms in fecal smears. Open feeders and watering vessels were used in the cage. This definitely demonstrated that the disease can be spread by fecal contamination of the food and water. When the older bird was killed on the 18th day it presented typical symptoms and lesions of blackhead. The liver, however, was only moderately involved and the ulcers were very small.

Chicks four days of age were fed fecal material from infested chickens and in eight days were, in some instances, passing the histomonas organisms in the feces, although the symptoms did not develop until later. Thirty-nine days after feeding the fecal material the control chicks averaged one pound in weight each while the histomonas infected chicks averaged 0.6 pound each.

These transmission experiments demonstrated the infectivity of fresh fecal material in perpetuating this disease once it has become established in a flock. The Van Es and Olney experiments showed that out-

Fig. 2. Liver of turkey that died of blackhead. Note that there are only two small areas of normal tissue in each lobe of this liver. Light areas indicate dense collections of embryonic cells due to action of virus

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door lots remain contaminated over the winter period. These investigators used turkeys in their work, however, and the transmission could have been accomplished by means of cecal worms.

Several broiler raisers have reported that a disease very similar to blackhead has been occurring on their farms for several years. In one instance a broiler producer, on whose farm the disease was endemic, reported that he had been feeding a neighbor's flock about two weeks when the disease manifested itself by heavy death losses in the second flock. This field demonstration illustrates the danger of spreading this disease by the attendant possibly carrying contaminated material on his feet.

Control Measures

Histomoniasis appears to be reasonably well controlled by the usual sanitary procedures, although in the outbreaks which have been investigated recently the use of sulphur and phenothiazine have also been used. McCullough and Nicholson[4], and Allen, Oliver and Peterson[5] have reported successful use of phenothiazine in removing cecal worms from poultry. The purpose in using phenothiazine in broilers has been to remove, if possible, the young cecal worms frequently found in the ceca of histomonas infested birds in order to prevent further damage to the ceca. The dose of phenothiazine recommended is 0.2 gm per broiler.

In controlled experiments conducted in the laboratory, the feeding of phenothiazine had no effect on either the number or the motility of histomonads voided in the feces of the treated birds.

The sulphur was recommended to prevent, if possible, the additional damage caused by a simultaneous infestation with coccidia before the death losses from histomoniasis stopped.

In those flocks where the owners have cleaned the houses thoroughly, removed all chickens showing symptoms and used approved feeders and watering vessels the losses from histomoniasis have been low.

Summary

1. Lesions similar to those found in histomonas infested turkeys have been found in broilers under farm conditions.

2. Organisms similar to *Histomonas meleagridis* have been demonstrated in cecal and hepatic tissue.

3. A disease simulating histomoniasis has been transmitted by pen contact of infected and normal chickens and by feeding fecal material from infected chicks to normal chicks.

4. Sanitary measures with the elimination of cecal worms and the prevention of coccidiosis appear to prevent the spread of histomoniasis within a flock.

5. Chickens experimentally infested with histomonas did not make as satisfactory growth as did the controls.

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[5] Allen, R. W., L. Oliver and H. O. Peterson, 1942. The efficacy of phenothiazine for the removal of the cecal worm of chickens. *Vet. Med.*, 37:10, pp. 412-15.

Poultry Perpetuates Tuberculosis In Livestock

Some time after the program to suppress and eradicate tuberculosis among cattle was inaugurated in 1917, it became obvious to live stock sanitarians that poultry were serving as a reservoir of the tuberculin infection for other animals, particularly swine and cattle.

Extended surveys have formed the basis for a reasonably accurate tabulation of the distribution and sectional prevalence of tuberculosis in farm poultry. They indicate that the greater prevalence of avian tuberculosis occurs in the north central states, an area which forms a large part of the region commonly referred to as the "corn belt."

This area of higher incidence of tuberculosis in poultry is the greatest egg and poultry meat producing section of our country.

A program for the control and eradication of tuberculosis in the poultry industry is desirable not only to benefit that industry but also to reduce condemnations of swine and beef carcasses.—*Excerpts from Indiana Poultry Blue Book.*

★ ★ ★ BUY WAR SAVINGS STAMPS AND BONDS

Autobiography of a Veterinary Practitioner

By E. T. BAKER
Moscow, Idaho

XIII

Other Factors in Practice

Ready for the Next Call

WHEN I return from a call, the first thing I do is to replenish all supplies used on the last trip. In winter, all aqueous solutions are kept in the house or cellar, and during cold weather, my emergency grip is kept in a back room where it is warm. I have often regretted our garage is not heated.

Having the equipment and car ready, all I have to do when the next call comes in is to dress in appropriate clothing, and drive out at moderate speed to my destination. There is no fuss or excitement, and no time is lost, which is important in emergency trips.

Between calls I prepare commonly used capsules, powders, lotions, liniments, ointments, bandages and other necessities, so that everything is ready when needed.

For years I have worn coveralls slipped over my everyday clothes. In winter, I wear a heavy sweater under the coveralls, and a good mackinaw over them. I always wear rubbers in summer, and four buckle high tops in winter. In this way, and by using aromatic antiseptics, I never "smell like a barn." I have a heater in the car, but seldom use it, as I find it better not to get out of a heated car and have to strip off in a cold barn, as is necessary in cases of dystocia or retained afterbirth. On long trips, where I know I am going to see a case of milk fever and the weather is really cold, I turn on the heater.

For some years, I did not realize the importance of suitable clothing, and, on account of this oversight, I ruined many a good pair of trousers or a clean shirt. Now, I keep all my old shirts, with the sleeves cut off, and keep my discarded pants, also. When I get a call for a dystocia or the like, I can wear these, and by changing at home, be ready for the job when I arrive. I carry rubberized trousers in the car, and use them when needed, but there are cooler outfits on a hot summer day than these.

One can do better work when he is suitably dressed for the occasion. He "does not hold back" for fear of ruining his clothes for he knows he can change later.

When I return home, I wash my hands well with tincture of green soap and warm water and, after drying my arms and hands, rub liquor antisepticus over them. This seems to remove all odors. Of course, whenever possible, I wear rubber gloves.

Although not a fussy dresser, I am a great believer in appearing clean and neat, but have never been much on wearing various uniforms, and resembling the chef in a hamburger stand. White robes are fine for angels, I am told.

Personal Hygiene

Nothing is more important to the successful practitioner than good health, as poor health renders him grouchy, disagreeable and often unable to do full justice to his work.

Perhaps, forty years ago it was a sign of greatness in one's profession to be gruff and snap his clients off and thus impress the proletariat with his independence, but

that day has disappeared. It is the pleasant, affable man who succeeds nowadays. The public cares nothing about the way "we may feel." They care nothing about our individual aches or pains. I have gone out on a call so weak from the effects of the flu that my son had to drive the car.

Excessive drinking or smoking, various infections, troubled home life, financial worries and a natural-born "sour-puss" disposition will ruin the best practice in the land. Sufficient sleep, nourishing food, comfortable surroundings and pleasant recreation are absolutely necessary to maintain good health.

Years ago, I figured my body was the only one I would ever have in this vale of tears and that I would try to take just as good care of it as I did of my car and equipment. When I am tired, I rest; when I am not hungry, I eat very little; when I feel like staying at home and listening to the radio, I do just that, and am never out late two nights in succession. I have never used tobacco, but I do not think the moderate use of it is harmful, and the same applies to liquor. Wild parties and imprudent excesses are what take a toll of our vitality which is not easily repaired. I do not believe in food fads, and am strong for good old "belly vittles." I have always been an early riser, and we have our meals on Sunday the same time as on week days.

For years, I have gargled my throat with liquor antisepticus, full strength, morning and night. Years ago, while working with "flu" among pigs, I contracted the same ailment and I have always been careful ever since. While driving an open car some twenty years ago, I was troubled with a conjunctivitis, with a slight pterygium, or thickening of the cornea. I now apply to the upper lid, night and morning, a little of the following solution: Camphor water and boric acid solution, of each one ounce; zinc sulphate, two grains. For a bad case of inflamed eyes, one can apply a little subiodide of bismuth with a small swab, or iodex ointment to the upper lid at night.

While performing autopsies, cleaning cows or working among infections, I always wear rubber gloves. If the infection is severe, I burn the gloves afterwards. I always disinfect my rubber shoes before leaving the premises. When I get home, I wash again, and use the liquor antisepticus, and, in cases like swine or equine influenza, use a nose spray. When I work around pea dust, I find washing my face and neck well with soap and water, and applying a facial lotion, will prevent any skin eruptions.

These few suggestions I have outlined for my own personal health take only a few minutes of time daily, but they have paid good dividends. I think if we conduct ourselves as professional men, we will be more highly thought of by our clients, friends and neighbors. A veterinarian will hold just as high an esteem among his community as any other person if he merits it.

No one, except the family physician, who receives money for listening to the complaints of chronic invalids who never die, gives a hoot about other people's various aches and pains. This reminds me of an old patient who was really a breadwinner for me. She was a big, determined looking Shorthorn cow who had milk fever every time she calved. When she was about dry, she would have a new owner, who would send for me in a hurry when she came down with milk fever again. This continued for five or six years, and I must have received around $75 for doctoring her. I began to have a great affection for her, and to figure on her to pay my national association dues each year. However, she became peevish in her old age, and kicked her last owner between the collar bone and the granary and, in a fit of anger, he sold her to the butcher. It may be compared to the young physician, who became associated with his father in practice, and, who one day proudly announced:

"Well, Dad, I finally cured old lady Greenback."

"Good God, Son, you haven't done that, have you?" shouted the old man. "Why, she sent you through college, boy."

A young veterinarian will locate in a town, and be so energetic and altruistic he will spend all night trying to keep disease down in his neighborhood. After doing this for a year or so, he moves to greener pas-

tures, somewhat sadder but about seven dollars wiser. Now, we are not advocating a careless attitude towards infection, but we have found it pays to let nature take its course and make a trip after one is summoned. County agents and extension veterinarians lie awake at night trying to keep down all disease and accidents in their territories, but we note about twelve thousand practitioners are still making a pretty fair living.

Dispensing

In the first place, I am on friendly terms with our druggists, for I buy from them nearly everything I use except specialties and biologics. Very few prescriptions are written, and they do not expect it, for most of our work is along emergency lines and we carry along our supplies.

When a client comes in for some medicine, it is always mixed up in front of him. He then feels he is getting individual attention. The practitioner who merely reaches up to a shelf and hands the client a package all ready made up, with a printed label, is only doing what any peddler can do. Of course, I have a few things ready for use, such as bloat and impaction mixtures; dusting powders; barbwire lotion in dauber bottles, and the like, but the great majority are mixed up as needed, including all mineral powders.

In winter, when the roads are blocked with snowdrifts or mud, many a call can be avoided by having the owner come in for medicine. Some of these cases have gone so long they will die anyway, and it is better to get one or two dollars, cash money, for some medicine than to make a dangerous trip and put another call on the books, perhaps forever.

I never dispense anything less than a dollar. If under that amount, I usually "throw it in," if to a good client. If a dairyman wants a few dilators, I give him a bottle of liniment to use on the quarter, and charge him a dollar for "the works." When a "kid" brings in a pup or kitten, I never charge anything unless it belongs to a well to do citizen, and, even then, if I like the kid, the service is not put on the books. This must pay dividends for, in all the twenty-four years we have lived on our corner, we have never had anything disturbed—even on Hallowe'en. Nothing pays better than having friends among the younger generation.

Probably enough medicine is dispensed to purchase all our supplies and, in addition, many clients come in for more medicine and to tell how the case is progressing for I seldom call up to find out. I can give no reason for this, except that I do not like to use the 'phone any more than is necessary. Another thing, never give a client too much medicine at one time. As mentioned before, he will keep it for another case "actin' like it," or loan it to his brother-in-law, and thus, in either case, you will be out a call.

In other words, our medicine room is the center of veterinary activities in our community. Dairymen know where the bloat medicine is kept, ready for use, and plainly labeled. If I happen to be out, or no one is at home—which seldom happens—they can rush in and get a bottle, for this room is never locked. I keep what few narcotics I use in a secluded place but have never had any trouble along that line.

A few days ago a man who raises over two hundred pigs a year came in for some worm medicine for about twenty head that were not doing well. Probably they had a little enteritis. I mixed up about five pounds of powder and charged him six dollars for it.

"I kin gitta sack of boughten worm powder fer half of that," he grinned, "but your stuff does the work." The reason it did was that I knew what was ailing his animals and prepared the medicine accordingly. He comes back again and again for similar medicine because he gets results and has confidence in the powder which is made up before him. I know what our community needs in the way of minerals and what kinds of feed are used and, consequently, can prepare medicines for each and every purpose. I think that is better than handing a client a sack or bucket of "Killam's Sure-Pop Mineral Powder," with a profit so small that when one client fails

or forgets to pay his bill it turns out that the entire shipment was handled for exercise only. As Doctor Riley of LaGrande, Oregon, expressed it: "I'm not in business for my health; I'm healthy enough."

Every now and then I take a gallon bottle and put in odds and ends of small quantities of medicines suitable for the occasion, and fill it up with red aromatic mineral oil. When a dead beat client comes in for a bottle of medicine for an ancient steed worth no more than the owner, I give him a bottle of this mixture, and he is satisfied. It does the animal just as much good as more expensive stuff, and is certainly easier on our pocketbook.

Mineral Handling

On account of my work on goiter, and various other diseases, I soon realized the lack of proper minerals in the soil, feed and water in many localities. However, there are other obscure conditions that must be taken into consideration, such as the kind of seasons, assimilability of the different elements, and various hereditary or pre-disposed idiosyncrasies of the animal iteslf. We all know of dairies, such as state college outfits, where the cows are given a balanced ration, with sufficient minerals, but one or two animals do not thrive. Why is this? It is not in the food given them, but rather some derangement of the body functions. To give these animals more "minerals" would only be "kidding the owner."

The original idea of various mineral containing powders, used under expert supervision, was fine and would have done a world of good. However, like many other things, this was abused, and many members of our own profession helped to bring this about.

My own observations lead me to believe that the promiscuous sale of minerals will wreck a practice, and it does this in two ways: First, the stock owner has such blind faith in its efficacy after listening to a high pressure salesman and feeding accordingly that he allows his animals to become hopelessly sick before calling in expert assistance. Not long ago, I was called to see a drove of nearly a hundred hogs in the last stages of swine erysipelas. The owner had purchased a large amount of a certain mineral powder and was giving it to the animals as per directions, and he "wondered why his mineral dope didn't straighten up them lame pigs?" He is out of my territory, but I did a little missionary work on him and I hope he knows more than he did. Colt owners think a mineral powder should eradicate bots, prevent navel ill and keep chicken lice off the foals. In winter, the owner of a horse will let an animal suffer for several days with impaction before summoning a veterinarian.

"Gosh, Doc, I bin feedin' that Bully Mineral Powder what says it prevents all kinds of colic and clogs," he says sadly, as I tell him he had better send for the undertaker.

The second reason, large scale mineral handling ruins a practice is that it tends to keep stock owners away from the local veterinarian. He begins to buy more and more "things outen a bucket," and feeds it indiscriminately to all his animals and poultry. The local practitioner twiddles his thumbs and, finally, moves to greener pastures.

Less than twenty years ago, three practitioners thrived in three small towns some thirty miles from me. They were in better live stock localities than I am but they began to "peddle minerals by the ton." They made a little easy, quick money. Now, the stock owners use these powders for everything. If an animal becomes sick, they let it die. One of the practitioners died; the others went into federal service. Those communities are ruined as locations for practice. Once in a while I receive a call over in that community for a milk fever or for some valuable animal, and I have a chance to observe conditions in these "ghost towns."

Now, I am not condemning the handling of minerals. That is for each individual practitioner to decide for himself. I am merely giving my views and observations and the fact that I am still in business at the old stand shows that some of my ideas are partially correct.

(To be continued)

Clinical Observations on Milk Fever

By LEROY E. BOWEN
Lynchburg, Virginia

FOR a good many years I have felt that some day I should like to express my views and observations from field experience about the old disease, milk fever and its complications. The more experience I have with its various manifestations the less satisfied I am with the present conceptions concerning it. It is true we know how to cure the majority of cases, but it would be interesting, from a scientific point of view, and it might be important, economically, if we could find out more about the cause. Ask almost any one, veterinarian or dairyman, what causes milk fever and he will say "calcium deficiency." Veterinary students are taught it and all agricultural papers and magazines agree. It is also said that milk fever is peculiar to heavy milking, fat, fresh cows. The latter statement at least is erroneous and one can but wonder at the unanimity with which it is chanted.

The Holstein cow, for example, is a breed that has held the world's record for milk production on several occasions. In the better herds they are heavy producers, well fed and fat—a classical set-up for milk fever if such teaching be true. Two fine herds of Holstein cows numbering about 60 each have been under my observation for 25 years. This observation represents probably 2500 calvings. In only one case, during this time, have I suspected milk fever. This was in a fresh cow, down in the field. I gave her calcium gluconate. She recovered. In my experience, not only with these two herds but in all herds in my district, the ailment is almost unknown in the Holstein breed. This week I treated a typical Holstein, as to size and color, for milk fever. She readily responded. However, inquiry elicited the information that she was half Holstein, and half Jersey. I don't say Holsteins do not have milk fever, only that after many years of practice in a fairly good dairy district, I have yet to see my first case of milk fever in a pure-bred cow of that breed. In my practice every black and white cow that has milk fever proves, when her breeding is revealed, to be 50% or more Jersey—less often some other cross.

A good many years ago there was an exhibit of a large number of fine Holstein cattle at a fair which I attended. They were making the fair circuit. Upon questioning the manager whether milk fever had given him any trouble, he stated that he had managed this particular herd for three years without a single case of the disease. This was what I expected to hear.

There are Guernsey herds in this vicinity in which milk fever occurs occasionally. But it is rather uncommon in this breed.

There are few, if any, Ayrshires in this community therefore, I can express no opinion as to their susceptibility to milk fever.

Pure-bred Jerseys and Jersey crosses are plentiful in this vicinity and among them 90% of our milk fever cases occur.

Referring again to the theory that a deficiency of calcium in the blood, or possibly in some other organ is the cause of milk fever; are we to assume that there is less calcium reserve in the Jersey and Jersey crosses than in the Holstein? If the answer is yes, it poses the question, why?

Let us consider the butter fat. In this respect the Holstein and Jersey breeds differ strikingly. Barnyard and hill-side experience arouses the speculation that the trouble may lie in that which is peculiar to the Jersey cow—an extraordinary production of butter fat for her size and capacity.

Several years ago, I was on a farm on some business, which I do not recall at present. The owner had a fine Jersey herd. He asked me to the barn to see two fine fresh cows of which he was extremely proud. When we got there, we found both down with milk fever, much to his surprise. This ailment has occurred on this farm, several times a year, for years without a single missed year—a glaring contrast to those two Holstein herds.

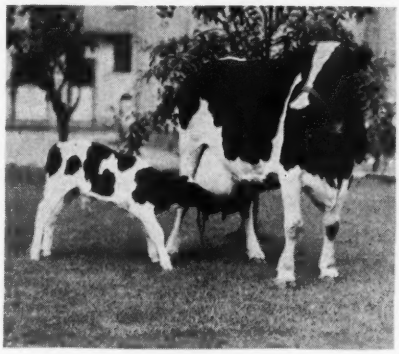
My father was a veterinarian and I can

remember when he almost refused to go to see milk fever cases because he didn't know what to do for them. Then in the 90's Schmidt of Kolding discovered that a small amount of potassium iodide in luke warm water, sterilized and infused into the udder cured many of his cases. Later it was discovered that inflation with air did just as well as infusion with potassium iodide solution. I recall the beginning and the end of the glucose deficiency theory; then came calcium chloride and headaches; then calcium gluconate in sterile water. "Put it in warm water and it will go back into solution and be usable." But sometimes, before it went back into solution, the cow died. So that, too, had to be discarded. Then came calcium gluconate with an agent that held it in solution in 250cc doses, the best yet, and as a treatment, near perfect. But what am I treating other than the name—milk fever? Recently I gave a Jersey cow nine injections of the gluconate and several udder inflations within a period of about three weeks. Eventually she recovered. Others have received one to eight treatments.

Other factors noted are: After a hard winter, which results in insufficient feed and thin cows in the spring, we have little or no milk fever, even in Jerseys; yet those conditions would appear to predispose to calcium deficiency. However, if spring brings plenty of rain and a good growth of grass the number of cases in the cows coming fresh later on in the summer is above normal. If the summer is dry and the fields are burned up and there is little pasturage, the ailment almost disappears. Again these conditions are just the reverse of what we should expect if a calcium deficiency were responsible for the trouble. Where we should expect it, it doesn't occur; where it should not occur it is prevalent. It seems that milk fever is a metabolic disturbance of unknown cause and that a mal-distribution of calcium in the body is just one of the effects, like slow respiration, inability to stand, etc.

In my experience, milk fever attacks cows that are approaching calving, calving or have calved recently. I have read for years and years various ideas about milk fever, complicated milk fever, acetonemia, grass tetany, etc., to little purpose. An editor's note in the November-December *Jen-Sal Journal* states: "The research studies on complicated milk fever by Greig of Scotland and Sjollema of Holland, coincide with the conclusion of the late P. A. Fish of New York that the etiology of complicated milk fever is still wrapped in mystery." I agree unreservedly with that statement.

The calcium theory, in my opinion, is a myth. Acetonemia and grass tetany are so closely related to milk fever that the practicing veterinarian can hardly differentiate between them, yet both seem to respond to any one of three treatments—udder inflation, calcium injections, or chloral hydrate. The udder inflation and chloral hydrate administration seem peculiar ways to increase the calcium content of the blood. Doctors E. T. Baker of Moscow, Idaho and S. L. Stewart[1] of Olathe, Kansas, both widely experienced practitioners, have also pointed out some vagaries in the manifestations of calcium deficiencies. They state that milk fever is far more prevalent in muggy, murky weather than it is on clear days. A low barometer forecasts not only unsettled weather but many calls to treat cases of milk fever; i.e., acute calcium deficiency. A high barometer is a portent of clearing weather, few calls to treat malk fever; i.e., waning calcium deficiency. These little peculiarities have been explained to me by some colleagues, but they don't take.



In my experience, milk fever is practically unknown in the Holstein, although a heavy milker

[1] Stewart, S. L., 1941. A practitioner's experience with milk fever. *Vet. Med.* 36:5, pp. 266-269.

Brachiocephalic Thrombosis and Aortic Arteriosclerosis in a Horse

Thrombosis and arteriosclerosis are described by Hutyra and Marek, and although these conditions are more rare in animals than in man, it is said that they may occur in any blood vessel in the body. Arteriosclerosis in a clinical case of Johne's disease was reported recently by Gifford, Eveleth, and Gifford.[1]

Thrombo-embolic colic in horses caused by the larvae of *Strongylus vulgaris* is described in most text books. This is probably the most usual form of thrombosis in large animals.

Arteriosclerosis in old dogs is apparently quite common, a most striking case with cartilage and bone and myeloid marrow in the aorta was found by Hueper.[2]

Aside from thrombosis of the anterior mesenteric artery in horses, the condition has been found also in the iliac artery,[3] posterior aorta,[4] and the vena cava.[5]

In the United States, the foregoing forms of circulatory disturbances in large animals are inadequately described in the literature and reference to them is seldom made. We have been unable to find a discussion of conditions similar to the one described here in the literature of this country, or specific reports of brachiocephalic thrombosis in foreign literature.

Case Report

November 20, 1942, an eight-year-old, chestnut saddle pony was brought to the veterinary clinic of the Kansas State College with a history of having been treated by two different veterinarians but having failed to show improvement. A diagnosis had not been made.

Symptoms.—The animal was thin and emaciated, the hair coat rough and the skin dry. The front limbs were enlarged and swollen, edema extending from the coronary band to the ventral surface of the thorax. All movement seemed to cause the animal pain in these members.

Depression and muscular incoordination were well marked. The horse was listless and showed little interest in its surroundings, examination or observers. It would eat and drink. The bowel action was normal, but at times it took a mouthful of hay, and stood with it hanging from the mouth, not chewing for a while, then suddenly made very forceful mastications. Other times while drinking, the pony would stop, and allow the muzzle to remain in the water for some time. It was impossible to back the animal from the stall.

The arrow points to the origin of the common brachiocephalic trunk. The X indicates the thrombus in the opened brachiocephalic trunk

The temperature was 102°F. and remained at this mark until death. Pulse and respirations were accelerated with frequent variations. The heart beat was pounding and jugular pulsation at times was strong. Waves of pulsation passed along that vein which were easily visible.

A fecal examination revealed an infestation of strongyles.

[1] Gifford, R., D. F. Eveleth and W. Gifford, 1942. Arteriosclerosis in a clinical case of bovine Johne's disease. Vet. Med., 37, pp. 416-420.

[2] Hueper, W. C., 1942. Cartilage and bone with myeloid marrow in the aorta of a dog. *Jnl. A.V.M.A.*, 101, pp. 493-94.

[3] Merillat, L. A., Thrombosis of the Iliacs, 1939. *Vet. Med.*, 34, p. 487.

[4] Daubard, 1939. Thèse, Lyon Abst., *Recuèil de Médecine Vétérinaire*, 115, p. 303.

[5] Tuxill, A. J., 1939. Thrombosis of vena cava in the horse. *Cor. Vet.*, 29, pp. 422-24.

After a few days the tissues of the fore limbs seemed to be breaking down, as indicated by a posterior flexion of both carpi.

The Blood Picture

November 23, 1942			December 9, 1942		
R.B.C.	5,700,000		R.B.C.	5,423,000	
W.B.C.	36,400		W.B.C.	41,700	
Hg.	8.5	(Dare)	Hg.	8	(Dare)
Stab.	35		Stab.	4	
Seg.	54		Seg.	74	
Bas.	1		Bas.	0	
Mono.	1		Mono.	3	
Juv.	0		Juv.	0	
Eosin.	0		Eosin.	0	
Lymphocytes Large	3		Lymphocytes Large	9	
Lymphocytes Atypical	6		Lymphocytes Atypical	2	
Lymphocytes Small	9		Lymphocytes Small	9	

Diagnosis and Prognosis.—Diagnosis was withheld, but an unfavorable prognosis was made.

Treatment.—Five grams of phenothiazine was administered *per os* daily for five days. Twenty-five minims of concentrated hydrochloric acid in 500cc of sterile water and 500cc of a 25% dextrose solution were given intravenously every 24 hours until the day death occurred.

Morbid Anatomy.—The animal died Dec. 10, 1942, and a necropsy was held at once.

The common brachiocephalic trunk was occluded by a well developed and well organized thrombus for the full length of the vessel, which in this case was about five centimeters. The thrombus was firm and adhered to the vessel wall, as it seemed to be included in both the intima and media. Canalization resembled the coiling of the nasal turbinate bones. Some blood, of course, was permitted to pass through the artery but due to the valve-like construction of the formation, circulation was difficult and almost completely obstructed. The vessel wall was somewhat thickened but there was no evidence of calcification.

The aorta was thickened and enlarged. When opened and laid flat it measured 16cm across at the heart, and was from one-half to one and one-fourth centimeters in thickness. Arteriosclerosis was evident for a distance of 36cm from the heart along the vessel; calcium having been deposited throughout the wall, some of it raised in whirls 2 to 5cm in diameter. There was no elasticity in this portion of the aorta.

There was hypertrophy of the left ventricle of the heart and the wall measured up to 11cm in thickness.

The remaining viscera was normal. No verminous aneurism was present in this specimen.

Valley Falls, Kans.

LESTER H. BURKERT

A Necropsy Absolves the Veterinarian from Blame

One evening during August, 1942, there was brought to Drs. Case and Planz of Akron Veterinary Hospital, a bitch which was having some difficulty in whelping. She had started to labor in the morning and, as yet, had not delivered any puppies.

After 30 minutes of observation it was obvious that assistance was needed to save the lives of the bitch and her litter. Obstetrical forceps were used, and a live puppy was delivered. For the next 30 minutes she was left unmolested in a quiet place with the hope that she might deliver the remaining puppies without aid. At the end of this time she was still laboring. More traction was used, and three more puppies were delivered in the next two hours.

The next morning the bitch and puppies were normal and were sent home. On the following morning the owner brought the bitch into the hospital and said that she died during the night. He seemed to blame her death to some bungling obstetrics or improper handling during her stay in the hospital.

After questioning the owner as to the treatment he had given her since she left the hospital, he admitted that she was very hungry the previous evening, and he gave her a large piece of cheese. With the owner's approval, in order to find out the exact cause of her death, an autopsy was made.

The uterus was examined very carefully and no signs of rupture or infection were evident, thus relieving obstetric manipulations from blame. In the course of examination of other organs a large piece of cheese was found in the glottis, extending one-third of the way down the trachea.

This large foreign body convinced even the owner that death was due to asphyxiation. Such an example reveals the value of a postmortem examination. It often corrects an erroneous idea of the client and gives the veterinarian "A Grand and glorious feeling."

MELVIN J. SWENSON

Concordia, Kans.

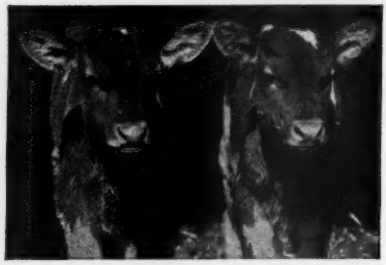
Paint Poisoning in a Calf

Lead poisoning is one of the most common forms of metallic poisoning in animals. This is due to the extensive use of the many lead compounds in agriculture. Cattle are very fond of licking paint. Fresh or old painted surfaces or discarded paint buckets offer a few of the common sources of "paint poisoning" on the farm.

"Of all domestic animals cattle are the most sensitive to lead poisoning in relation to their size, calves being especially susceptible."[1]

"In acute lead poisoning there are two distinct groups of symptoms: gastroenteritis due to the caustic action of the chemical upon the mucosa, and cerebral symptoms arising from the effect of the lead upon the nervous system. Cattle often manifest excitation of consciousness and motor irritation and these are of great assistance in making a diagnosis. The mortality in acute lead poisoning is high, often 100%. I have never observed a recovery in a calf. When only a small amount has been consumed by mature cattle recovery is possible."[2]

In this case a four-week-old Guernsey heifer had licked approximately sixteen square inches of paint from the inside wall near which it was tied. The wall had not been painted in the last ten years; however, it still offered a substantial source of basic



The owner had already lost several calves from paint poisoning

lead carbonate. Dr. L. W. Mick, Akron, Ohio, was making pregnancy and sterility examinations when the calf began showing marked symptoms of mania, followed by convulsions. Thus the first symptoms observed consisted of derangement of the central nervous system. The owner had lost three calves the week before from "paint poisoning" and was quite sure the animal would die as the others did not live long after going into convulsions.

Doctor Mick administered 30cc of a 20% solution of magnesium sulphate introvenously. The calf was in a state of quiescence for several minutes, followed by another convulsion. Immediately an additional 30cc was administered intravenously and 60cc subcutaneously. Two ounces of magnesium sulphate in several quarts of water was given *via* stomach tube, also.

Two weeks later, while attending another case at the same dairy, this calf was seen again. At that time it was alert and in good condition. Had veterinary service not been available when the calf showed the first symptoms of lead poisoning it probably would have died.

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[1] Giltner, L. T., 1942. Yearbook, U. S. Department of Agriculture, p. 643.

[2] Udall, D. H., 1941. The Practice of Veterinary Medicine, 3rd Edition, pp. 638-39.

Abstracts

Carotene Content in Relation to Vitamin A Deficiency

Periodic determination of the plasma carotene level[10] of groups of cows receiving prairie hay as their major source of carotene gave evidence that those on a full hay ration varied from 250 to 479 μg. of carotene per 100cc of plasma, those on a 50% normal hay intake from 179 to 201 μg., and those on a 25% normal level from 80 to 167 μg. Cows whose plasma carotene during the last 90 days before calving averaged less than 150 μg. per 100cc were generally in poor condition after calving, and many of the calves were weak and failed to survive. Apparently this level of plasma carotene indicates a border-line deficiency of vitamin A.

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Poisoning of Turkey Poults from Whorled Milkweed (Asclepias Galioides)

A review of the literature on milkweed poisoning shows that various species of this plant are known to be toxic for horses, cattle, sheep, goats and in a few instances for chickens.[12] The earliest record of sus- Pammel (1917), who reported the loss of about 500 chicks, during one season, which fed on young sprouts of Asclepiadacae in New Hampshire. In southern Colorado during the spring of 1941, a turkey raiser turned a few early hen-hatched poults, three weeks old, into a small yard where young milkweed plants were growing. Soon afterwards several poults had a series of convulsions and died within a few hours. The camp of another flock of 950 half-grown turkeys was moved to an area on the river bottom where the vegetation consisted of cottonwood trees, willows, tough salt grass and scattered patches of milkweed. The latter was the only succulent green plant growing there. The following pected poisoning in chickens is given by morning 39 turkeys were found dead and 15 were sick. The writer saw one of these sick birds a few days later and it was still staggering and lacked coordination of its legs and wings. Nearly all died shortly.

In acutely fatal cases of milkweed poisoning of turkey poults, the first symptoms usually occurred within approximately one hour after the birds ate the plant and death came a few hours later. The prodromal stage was ushered in by drowsiness and listlessness, slightly irregular movements, and lack of coordination. When convulsions began, the fowl lost muscular control of its legs and wings, fell backward, lay on its side or even on its back, with wings flapping and legs trembling. These symptoms were of the clonic or intermittent type. The head was twisted and neck arched. Breathing became difficult and the bird was unable to stand. The pupils of the eyes were moderately dilated when compared with normal specimens. Before death occurred, the spasms became less in number and intensity, until finally exhaustion and coma followed. Increased embarrassment of respiration, accompanied by gasping, was noted. Victims of milkweed poisoning die from respiratory paralysis. The symptoms as above noted may be less severe in cases of a milder degree of poisoning, extending over longer periods of time. The temperature chart curve is generally downward.

The gross examination of poults which died within a few hours after being fed the milkweed showed only slight lesions. Their crops were generally distended with a small amount of gas and usually contained a considerable quantity of the poisonous plant mixed with mucus. The heart and mesenteric arterioles appeared congested. The lungs were slightly edematous. Histopathological sections of the brain, heart, liver and kidneys did not reveal significant changes.

The milkweed in question, *Asclepias galioides,* has a limited geographical range, being reported in western and southern Colorado, western Kansas, northern New Mexico, western and eastern Arizona, and in southern Utah. It is possible that this plant may also be found in northwest Oklahoma.

[10] Gallup, W. D., and A. H. Kuhlman, 1941, Carotene content of the blood plasma of dairy cattle in relation to vitamin A deficiency. *Okla. Acad. Sci.* Proc., 21, pp. 89-92.

[12] Stiles, G. W. 1942. Poisoning of Turkey Poults from Whorled Milkweed (Asclepias galioides). *Poultry Sci.* 21:3.

In regions where *Asclepias galioides* abounds, poisoning of turkeys may be of considerable economic importance. Adequate care should be exercised to prevent livestock poisoning in areas known to be infested by this plant. Sufficient wholesome green food should be provided for fowls.

The Pantothenic Acid Requirement of Chicks

The first reports of chick dermatosis, a syndrome characterized by granular sticky eyelids, crustations at the angles of the mandibles, and thickened fissured skin on the bottoms of the feet, were made in 1930 and 1931. The investigators used two diets in their studies, one containing raw egg-white as the chief source of protein and the other containing purified casein. Other workers showed that the severity of the dermatosis in chicks fed a purified casein diet is greatly increased by subjecting the diet to prolonged heating at high temperature in a dry atmosphere. Still other workers reported that the factor which prevents the dermatosis in chicks, fed a heat-treated purified casein diet is not identical with the one which prevents the dermatosis in chicks fed a raw egg-white diet. This finding has been confirmed[10] and others have demonstrated that the factor which prevents the dermatosis in chicks fed a heat-treated purified casein diet is pantothenic acid, while more recently evidence has been presented showing that the factor which prevents the dermatosis in chicks fed a raw egg-white diet is biotin.

Studies were undertaken to determine the quantitative requirement of chicks for pantothenic acid and the amount present in feedstuffs, and thus to disclose whether or not the dermatosis occurring in the field is identical with that resulting from pantothenic acid deficiency.

Under the conditions of the experiment, single comb White Leghorn chicks require from 500 to 550 micrograms of pantothenic acid per 100 grams of diet for the prevention of dermatosis and approximately 600 micrograms per 100 grams for maximum growth. In one experiment, Rhode Island Red chicks were found to be less susceptible to pantothenic acid deficiency than single comb White Leghorns and their requirement to be approximately 75 micrograms less per 100 grams of diet. These requirements are not in agreement with the requirement previously reported. By means of the microbiological assay and by calculation from the average pantothenic acid values of feedstuffs, normal chick diets were shown to contain from 1,300 to 1,700 micrograms of pantothenic acid per 100 grams.

Treatment of Chronic Mastitis

Seldom does *Streptococcus agalactiae*,[20] the most common cause of mastitis, appear in an acute form; although usually of a chronic nature *Str. dysgalactiae, Str. uberis* and others may be responsible for acute infections. The acridine derivatives, entozon, acriflavine, and also trypaflavin, administered by infusion. When an oil solution is injected via the teat canal directly into the milk cistern, advantages in large-scale work are smaller volumes of solution, less equipment required and simplification of instrument sterilization. Gramicidin or tyrothricin and novoxil have been used. Chemotherapy is a surgical procedure and should be conducted by a veterinarian, lest great damage to the udder result from careless technic. A conservative figure for cures in an average herd by means of udder chemotherapy might be 50%, with some herds going as high as 80 to 90% cures. Mild cases offer the best chance of success. Chemotherapy should not be used on acute cases. Tyrothricin and novoxil may be injected safely during the dry period and this saves material. Of the bactericidal agents effective against *Str. agalactiae* the author has found the alcoholic solution of gramicidin in sterile water and mineral oil, with the dose properly adjusted, was the least irritating and most effective.—E. F. Goss in *Bio. Absts.*

[10] Baurenfeind, J. C., L. C. Norris and G. F. Heuser, 1942. The Pantothenic Acid Requirement of Chicks. *Poultry Sci.*

[20] Little, Ralph B., 1942. The treatment of chronic streptococcic mastitis with various bactericidal agents. *Bul. Internat. Ass'n Milk Dealers.* 34:16, pp. 345-356.